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GASTROJEJUNOSTOMY: A REVIEW OF 100 CASES

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Of all the chronic ailments met with in Barisal, dyspepsia is the most common and of dyspepsia peptic ulcer constitutes the vast majority. Nobody knows why. But these patients have been found to have four important factors in common. These are economic condition, mechanical and chemical trauma, focal sepsis and nervous influences. The poorer classes of people in whom gastric ulcer is more common in Barisal, possess neither the means nor the knowledge to obtain proper food. Their diet consists mainly of carbohydrate. They can barely afford to have two meals a day which are often stale. They are accustomed to assault their stomach by bolting food without allowing time for mastication. They have one vice in common that is tobacco which often is smoked in excess and in empty stomach. These indulgences cannot affect harmfully 90 per cent of stomachs with a normal motility and secretion. It is in the remaining 10 per cent with hypersthenic diathesis that gastritis is set up and ulcers follow (Hurst). There is a tendency to incline more and more to the view that gastric ulcer is not a primary condition but it is due to an infection, perhaps from some distant focus and that the ulcer develops as the result of a septic embolus which lodges in a small gastric radicle leading to local necrosis of the mucosa. The resulting ulcer is prevented from healing owing to the irritation by the gastric juice and spasm of the underlying musculature. The ulcer cases of our series had pyorrhœa alveolaris in common. Local treatment and autovaccine from the pyorrhœa pus did a lot of good in some of our cases. Over-work, anxiety and all that are also common among these patients. The ulcer patients are not neurotic in the accepted sense; on the contrary they are careless in their habits and often capable of intensive activity over long periods. Cushing relates ulcer formation to abnormal parasympathetic discharges and vagotonia which results in hyperchlorhydria, hæmorrhage and ulcer formation. It seems, therefore, probable that the dual nerve supply to the stomach represents a balanced and mutually antagonistic mechanism so that highly strung people who are inclined to vagotonia are particularly prone to ulcers.

The classical syndrome of pain, vomiting and hæmorrhage in gastric ulcer and hunger pain and melæna without hæmatemesis in duodenal ulcer were often absent from our cases. In about 75 per cent of our series of hundred cases of peptic ulcer, the ulcers were characterised by hyperchlorhydria (regurgitation, pyrosis, burning and gastric pains alleviated by alkali or by food) and by the intermittency of the attacks, *i.e.*, periods of ill health followed by complete or relative good health. Ulcers are extremely frequent in Barisal, especially in men and duodenal ulcer is much more common than gastric. Of ten patients of gastropathy, eight cases have affection of that organ. The positive signs in ulcer of the lesser curvature was furnished by radiology (a niche, fixed and constant biloculation). In duodenal ulcer only in a very few cases were discovered distinct radiological signs (niche or lacuna from periduodenitis). The diagnosis of duodenal ulcer was made especially by fractional test meals. The distinction between pyloric, suprapyloric and

subpyloric ulcer was, however, more often not possible.

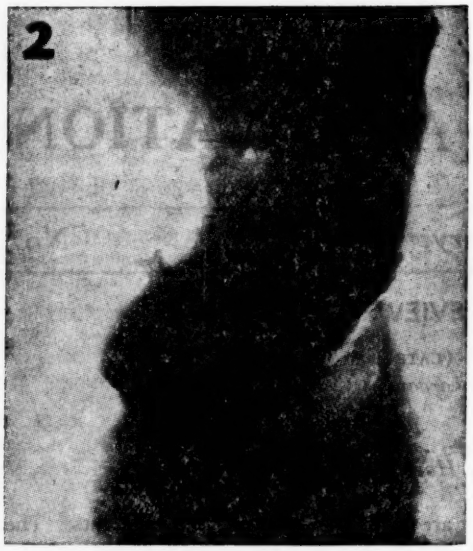

FIG. 1—Showing stomach dilated by obstruction and with feeble peristalsis.

The medicinal treatment of peptic ulcer has been much read and written upon. But can ulcers be treated medically? Yes, if recent; no, if the patient is over forty—after forty, one should fear, in chronic ulcer, cancerous change; no, if it be chronic and it is chronic, if the symptoms be intermittent or have been permanent for long. Recent ulcers being limited to mucosa may cicatrise from medical treatment. Cure is maintained by proper dieting and good general hygiene. Chronic ulcer resists medical treatment. During the periods of quiescence, the ulcer is still active though there are no symptoms. Essentially, medical treatment of ulcer means rest in bed, graduated diet extending over 4 to 8 weeks, continuous alkalization, and hygiene and strict discipline as to these points is insisted upon. As a rule, we institute medical treatment irrespective of the stage and age of the ulcer. With strict discipline, good results have been obtained in a third of the total number of cases. But these are all comparatively early cases. Transition from rest in bed to walking about extends over two weeks. Some cases become free of symptoms when a return to the ordinary fare is begun. We have not met with marvellous success with medicinal treatment of chronic gastroduodenal ulcers which are more than 5 years old. The ratio of failures of medical treatment in ulcer has been reported all the way from 1 to 22 per cent. Failures particularly occur in ulcers which are very chronic and in our series all the cases were old and extended over 5 years or more. It is our experience that a true chronic ulcer is generally a surgical condition and most gratifying results have been observed with surgical interference.

Before a case is admitted into hospital it is always explained to him that surgical intervention may be necessary at any time. Some cases prefer the operative risk to prolonged medical treatment. They are operated on immediately after the diagnosis is established. Even after the bed and diet treatment, the possibility of operation is mentioned to the patient. Should the symptoms appear during the bed treatment or there is a return of the pain or if there is no amelioration of the local symptoms, operative treatment is advised at once and carried out as offering the patient almost certain relief with the prospect of cure in over 95 per cent of cases at a very slight or no risk. In course of medical treatment any change in the character of symptoms is noted, such as loss of periodicity and gradual persistence of the pain or recent occurrence of evening vomiting. Such symptoms suggest developing pyloric stenosis and x-ray examination reveals the more prominent ulcer crater or cicratrical deformity of the stomach and 6 or more hours' retention. No time should be spent in operating on such cases. Ulcers left to themselves cause the patient risks: acute or chronic perforation, abundant or repeated hæmorrhages, pyloric or median stenosis, adhesive or suppurative perigastritis and cancerous degeneration in gastric ulcer. Independently of these risks what requires especially surgical interference, is the chronic dyspepsia, of which the patient is tired; it is the lessened vitality and the malnutrition which makes him an easy prey to a superadded infection, such as tuberculosis. Unknowingly it diminishes the vital, social, intellectual and moral efficiency of the patient, who is not considered ill, but a neurasthenic, a neurotic, a dyspeptic, a rheumatic, etc.

Operative treatment can be considered from 4 standpoints:

(1) Direct treatment by treating the ulcer locally by excision, cauterisation or burying has been followed by recurrence in 40 per cent cases at the Mayo Clinic.

(2) Indirect treatment by gastrojejunostomy. The new opening serves the four-fold function of giving rest to the ulcers and relieving the spasm by short circuiting the passage of food, affording better drainage of the stomach in pyloric stenosis and of allowing the alkaline duodenal juice to mix freely with the stomach contents so that the free acid secreted is permanently neutralised or reduced to small amounts only. The cases

which are most suitable for gastrojejunostomy are small and free ulcers at the pylorus. Pyloric stenosis, in addition, fully justifies this method of operation.

(3) Direct and indirect treatment combined. When the ulcer is not big and is adherent it should be excised, cauterised or buried and gastrojejunostomy performed.

(4) Partial or limited forms of gastrectomy has for its aim the removal of the ulcer together with a greater part of the ulcer-bearing area of the stomach.

FIG. 2—Stomach showing active peristalsis and 'Niche and Notch'.

It is stated that in the surgical treatment of gastric ulcer, gastrectomy should be the rule, other methods the exception. There is no doubt that partial gastrectomy is the only satisfactory operation for a gastric ulcer. But gastrojejunostomy is the best operation for duodenal ulcer producing stenosis. Partial gastrectomy has recently been highly recommended as the operation of choice for duodenal ulcer as well. But the free acid secreted in the stomach is permanently neutralised or reduced to small amounts only in 50 per cent of cases of gastrectomy done for duodenal ulcer in contrast with almost 100 per cent when performed for gastric ulcer. An anastomotic ulcer develops in the remaining 50 per cent with as much frequency as after gastroenterostomy. Further surgery is almost impossible for an anastomotic ulcer after gastrectomy. Besides, mortality of gastrectomy is considerably higher than that of gastroenterostomy. The cases under review, coming from the poorer classes and suffering for long periods as they did, were very much debilitated and gastrectomy was out of the question. With a patient with poor powers of resistance one ought to perform gastroenterostomy only and not fear to intervene again some months or years later, if the symptoms persist or return. There should be no hesitation to run the risk of operating on a patient twice to avoid death from one operation. Better to cure a patient by two operations than to kill him by one. Secondary gastrectomy is easy and without risk, if the anastomosis (jejunal ulcer) has not to be resected. As a rule chronic ulcers are attended with adhesions and engorgement. The only feasible operation for such cases is, in our opinion, gastrojejunostomy combined with local treatment of the ulcer. The undoubted indications of gastrectomy are large, adherent and penetrating ulcers and ulcers in which malignancy is suspected.

We performed in the District Hospital, Barisal, 99 cases of gastrojejunostomy and one case of gastrectomy. Out of the 100 cases of gastrojejunostomy, 6 had cholecystectomy as well for associated chronic cholecystitis. Three-fourths of the cases were Muslims and 95 were males and 5 females. All of them had a long history of pain for more than 5 years and 58 of them had suffered for 10 to 20 years. In our series, duodenal ulcer was found twice as often as gastric ulcer, and in 80 out of 100 cases there was definite pyloric stenosis.

Fractional test meal showed hyperchlorhydria in 75 per cent of our cases. About 15 per cent of the cases had no pain for years before admission into hospital. Their chief complaint was vomiting and x-ray revealed greatly dilated stomach and deformed duodenal cap or pylorus. In these cases occult blood was never detected at repeated examinations of the stool after admission into hospital. These were really cases of healed ulcer resulting in cicatricial contraction in the process of healing and were ideal ones for gastrojejunostomy. The cases in our series fell into three classes:

1. Nonstenosing peptic ulcers resistant to medical treatment (19 cases). These were treated by gastrojejunostomy alone.

2. Ulcers showing definite pyloric stenosis (80 cases). These were treated by infolding the ulcer and gastrojejunostomy. 6 cases had cholecystectomy as well.

3. Ulcer-cancer (1 case). This case was treated by partial gastrectomy of the type of Polya.

PRECAUTIONS BEFORE, DURING AND AFTER THE OPERATION

Before operation—Operation on the stomach requires: (*i*) A precise history and a complete clinical examination; (*ii*) examination of blood—Hb, R.B.C., W.B.C., Kahn and azotæmia; (*iii*) examination of urine

for acidosis, albumin and sugar; (*iv*) fractional test meals; (*v*) examination of stool for occult blood; (*vi*) a complete radiological dossier which tells one the probability of cancer or ulcer and their site. This dossier includes the radiological details of the intestine also, because too often the gastric symptoms are of reflex origin from cholecystitis, appendicitis or Lane's disease.

All possible sources of infection are eliminated. Postoperative pulmonary complications are prevented by disinfecting the nose, the throat and the teeth (previous removal of tartar and painting the gums with iodine). Respiratory gymnastics are a useful adjunct. The patient is taught to breathe deeply before and after the operation. This helps by oxygenating the blood and stimulating the patient's vitality. The lung is not only a chamber of air for the purpose of hæmatosis but also an internal secretary gland; deep breathing increases this function and consequently, that of all the endocrine organs. Veritable resurrections have been seen in patients who without this respiratory training would not have been fit for prolonged surgical interference. Diet and bowels are carefully regulated. Bowels are opened by saline enema daily for many consecutive days, specially if barium has been given. Purgatives are discountenanced in any case whatsoever. The patients being debilitated by long malnutrition are given intravenous glucose for a few days before operation. Cases with pyloric stenosis have rectal feeding with glucose saline and those without it are given plenty of liquid, fruit juice and alkalies. The stomach is washed out with alkali (sodii bicarb one dram to a pint) for a couple of days before operation. This helps to disinfect the stomach and to accustom the patient to the introduction of the stomach tube which is necessary in two-thirds of the cases after operation, because nausea, vomiting, fever, etc., result.

During operation—Though a few cases had been done under local anæsthesia (novocaine 1 per cent) combined with spinal (Neocaine 0·15 gm.) or splanchnic (novocaine 5 per cent) anæsthesia, we prefer general anæsthesia and in the absence of gas and oxygen, the common A.C.E. mixture is our choice. The abdomen is opened by a right rectus incision and a posterior no-loop gastrojejunostomy is performed as usual. It is very important to see that the stoma is not less than 2 inches in length, that the opening is at the most dependent part of the stomach both in the standing and recumbent position, and that no loop is left between the point of anastomosis and the duodenojejunal junction. Unless these precautions are taken secondary ulcer, regurgitant vomiting and intestinal obstruction are likely to occur. The ulcer which is recognised by a rough, red, stippled and puckered area and firm indurated feel is infolded and a tag of omentum grafted to it. At the beginning of the operation the abdomen is explored. The appendix, the gall bladder, the end of the ileum, and the pancreas are seen and palpated. The appendix is removed as a routine and in 6 cases the gall bladder was definitely diseased and removed. In one case cholecystectomy and gastrojejunostomy were performed in two stages, the patient being very feeble. In every case the boldness of the operation was strictly adapted to the patient's power of resistance. It is better for a patient to lose a month than to lose his life. In every case we give 5 per cent glucose in normal saline one pint, intravenously by drip method during operation.

After operation—This patient is propped up as soon as he recovers completely from anæsthesia. The patient must not suffer from pain whilst breathing, coughing, spitting, moving etc. Pain exhausts the patient's nerve and renders him prone to acidosis and pulmonary complications. Morphine is unhesitatingly given and repeated as often as is necessary. It is better to coat it with atropine which quietens the colic due to circulating gas in the intestines. Disinfection of the mouth is essential to prevent stomatitis and parotitis. When he asks for it as much sterile water may be given as he desires. On principle, after operation for gastric ulcer the later and the less the patient eats the better. He is ready for food in about 48 hours. But food should be increased very cautiously. We give thin barley, thin Horlicks, barley plus milk, milk and bread, milk and soft rice and rice and vegetable stew on successive days. For 48 hours after operation intravenous glucose 25 c.c. daily and rectal saline with 5 per cent glucose by drip method are given. The classical alkaline treatment is, we have found, unnecessary after operation. Some cases may even need acids such as acid hydrochloric dil 10 to 15 minims, three times a day. Respiratory gymnastics are encouraged. Immediately after the operation if CO_2 is administered, it helps in excreting anæsthetics from the lungs by deep breathing and prevents postoperative pulmonary complications. The stomach is regularly washed out in every case, the evening of the operation and repeated in cases of vomiting, fever, dilatation of the stomach, irrespective of the reluctance and supposed exhaustion of the patient. We never regretted having washed out a stomach too early.

Warm normal saline is used. For distension, if there be any, ice is applied on the abdomen and wonderful results have been obtained with injection of pituitrin or

prostigmine (Roche) and massaging the site of injection, while a flatus tube is in the rectum. The convalescence with the above procedure is more or less uneventful. Stitches are removed on the 8th day and excepting 3 cases all had primary union. Septic infection was rare in our series probably due to purer air of mofussil stations. The patient is allowed to get up after 10 days. Average stay after operation is 14 days. The patient is discharged with caution against over-eating and with advice to report his condition from time to time. A diet is prescribed for him to last for a year, albuminoids being forbidden. A little olive oil is advised with meals. One of the cases under review had an attack of cerebral thrombosis involving the right limbs but no cause could be ascertained. He, however, recovered to a great extent after prolonged treatment.

All our cases survived operation. Out of our series of 100 cases, 95 cases are immensely relieved and are doing well for the last year and a half or more. Only 5 cases have reported themselves with vague symptoms of hyperchlorhydria. 2 of these cases of apparent failure were readmitted and kept under observation. Put under strict regime of diet and rest they were relieved of their vague gastric pains and acidity. It is needless to say that these poor people can seldom afford to restrict themselves to the one-year-diet prescribed for them after operation. All these cases are still being followed up and if they remain free from symptoms for 3 years after operation, doing gastrojejunostomies, the mildest operation for peptic ulcer, instead of gastrectomies (involving a high percentage of deaths) will be amply justified.

A surprising change results immediately from the establishment of the stoma. Whether it is due to the relief of the delay in emptying the stomach or to the alkalizing of the acid gastric contents, pyloric spasm and hyperperistalsis are reduced or abolished and the patient at once loses the characteristic pain from which he had been suffering for years. That this cannot be due entirely to drainage is evident when we remember that in duodenal ulcer the stomach usually empties more rapidly than a normal stomach, and in many cases of gastric ulcer stasis is not present. But whatever the physiological process, the effect of the reduction of the gastric hypermotility is to place the stomach in a condition of relative rest, which is perhaps more important than anything else as an adjuvant in the healing of an ulcer.

FIG. 3—Skiagram one year after gastrojejunostomy. Plate immediately after intake of barium-meal which is seen filling up the jejunum through the anastomotic opening.

TABLE SHOWING THE ANALYSIS OF THE 100 CASES OPERATED

Age Group	*No.*	*Sex*		*Religion*			*Duration of illness in yrs.*	*Diagnosis confirmed by operation*		*Duration of stay after operation in days*	*Remarks*
		Male	Female	Hindu	Muslim	Christian		Gastric ulcer	Duodenal ulcer		
21-25	18	17	1	3	14	1	5-8	6	12	13-16	nil
26-30	19	16	3	3	16	—	5-10	7	12	14-19	One had cholecystectomy also
31-35	25	25	—	6	19	—	8-10	9	16	12-20	One had cerebral thrombosis; two had cholecystectomy also
36-40*	15	15	—	5	10	—	8-12	6	9	13-15	One had cystectomy
41-50	21	20	1	7	14	—	10-21	6	14	13-16	Two had cholecystectomy
									1 (ulcer cancer)	30	Had gastrectomy
Above 50*	2	2	—	—	2	—	6		2	22-28	nil

* One case of age group 36-40 and both cases of age group above 50 had stitch infection.
N.B. All the cases operated were cured.

COMPLICATIONS OF TONSILLECTOMY

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In the present paper a study of the incidence of the various complications, as met with in our hospital during a period of ten years (1931-40), is made. The tonsillectomies were done both in the out-patients and in-patients departments, by several operators. Some were removed by the guillotine, others dissected out, some were done under general anæsthetics, chiefly chloroform, while in some, local anæsthesia was employed. The series, therefore, represents a variety of cases dealt with under a variety of circumstances. The total number of cases so reviewed is 2,078.

HAEMORRHAGE

Undoubtedly, posttonsillectomy hæmorrhage of varying degrees of severity is the commonest and the most annoying complication. Loeb (1932) has remarked, "that, in general it may be said that moderate hæmorrhage is common, serious hæmorrhage not unusual, dangerous hæmorrhage may occur, and fatal hæmorrhage is rare." In our series we had 23 cases of hæmorrhage—mild, severe and dangerous, an incidence of 0·8 per cent. Sir Lancelot Berrington-Ward (1940) in publishing a report on the study of 1,078 consecutive cases of tonsillectomy mentioned 8 cases of reactionary hæmorrhage, one of them severe enough to need ligature of the bleeding points. His series also showed an incidence of about 0·8 per cent. Keen (1931) in reviewing 9,334 tonsillectomies, reports that the incidence of hæmorrhage is 1 per cent and that severe primary hæmorrhage depends upon the method of operation. I would, however, qualify this remark by saying that it depends on the operator rather than the method. Some surgeons have experienced a higher incidence of hæmorrhage. Brown-Kelly (1921) quoted 10 cases of posttonsillectomy hæmorrhage, scissors being used in one and guillotine in nine. He also quoted that Breaman Douglas had encountered 6·5 per cent cases of bleeding out of a series of 1,560 cases of tonsillectomies and there were 4 fatalities in 3 years. Irwin Moore (1928) came across one case with alarming hæmorrhage and quoted that Panse reported one case of fatal hæmorrhage in an adult of 17 years with cardiac disease. As a fact of historical interest he wrote that Gelsus (A.D. 10) who performed tonsillectomy by means of finger dissection alone, did not mention of hæmorrhage after tonsillectomy.

As regards the age of the patients it is a common experience that in children this complication is much less.

The following cases of hæmorrhage after tonsillectomy are cited:

Case 1—J. N. A., 22 years, male, operation under local anæsthesia; 2 ligatures at the time of operation. Hæmorrhage 2 days later from one tonsillar bed, controlled by application of Whitehead's varnish and pressure.

Case 2—C. P., 30 years, male, prolonged hæmorrhage, occurring soon after operation and controlled by intravenous injection of calcium, hæmostatic serum and local pressure.

Case 3—J. P. S., 23 years, male, very much adherent tonsils, operation under local anæsthesia; hæmorrhage from right side 3 days later, easily controlled by pressure and calcium given intravenously.

Case 4—Mrs. K., 25 years, female, tonsillectomy under local anæsthesia; some reactionary hæmorrhage same day; easily controlled by calcium and hæmostatic serum injection.

Case 5—W. K., 23 years, male, tonsillectomy under local anæsthesia; severe reactionary hæmorrhage—bleeding being controlled by ligating the bleeding points.

Case 6—S. H., 22 years, operation under local anæsthesia; hæmorrhage 1 week later, controlled by intravenous calcium and ligature of bleeding points.

Case 7—S. N. B., 22 years, male, operation under local anæsthesia; hæmorrhage 8 days later, controlled by ligature of bleeding points.

Case 8—B., 30 years, female, operation under chloroform anæsthesia; severe hæmorrhage at the time of operation; pillars had to be ligatured.

Case 9—J., 11 years, female, primary hæmorrhage; stopped by pressure and cold pack round neck in about 4 hours.

Case 10—Dr. B., 21 years, male, copious primary hæmorrhage, controlled by ligaturing pillars.

Case 11—A. C., 20 years, male, hæmorrhage 12 days after operation controlled by application of Whitehead's varnish and intravenous calcium.

Case 12—M. A., 22 years, male, operation under chloroform anæsthesia; hæmorrhage 3 hours after operation, the ligature of the faucial pillars did not stop the bleeding; the right external carotid artery was ligatured but bleeding still continued; blood transfusion was given and hæmorrhage was controlled.,

Case 13—D., 20 years, male, hæmorrhage 7 days after operation, controlled by application of pressure and cold pack round necks.

Case 14—S. B. K., 14 years, male, operation under chloroform anæsthesia, hæmorrhage on 7th day, controlled by injection of hæmostatic serum, local pressure and cold pack.

Case 15—M., 22 years, male, copious primary hæmorrhage controlled by ligature of the bleeding points.

Case 16—A. P. C., 10 years, male, operation under chloroform, hæmorrhage started on the second day; controlled by intravenous calcium and application of cold pack round the neck.

Case 17—M. U., 20 years, male, operation under general anæsthesia; primary hæmorrhage stopped by cold compress and hæmostatic serum injection.

Case 18—R. I., 20 years, female, hæmorrhage started on the second day after the operation under general anæsthesia, controlled by injection of hæmostatic serum and cold compress used locally.

Case 19—P. S., 20 years, male, tonsillectomy under local anæsthesia; hæmorrhage on the second day, controlled by suturing of the pillars.

Case 20—G. D., 20 years, male, operation under local anæsthesia; hæmorrhage started on the fifth day, pillars sutured together with no effect when external carotid artery was ligatured and viper venom was applied locally.

Case 21—C. R., 12 years, male, operation under general anæsthesia; considerable primary hæmorrhage, controlled by injection of hæmoplastin and local pressure.

Case 22—S., 12 years, female, primary hæmorrhage, controlled by pressure and application of Whitehead's varnish.

Analysis of these 23 cases shows that there were 11 cases of primary hæmorhage and 12 of secondary type, 4 were children under 14 years of age and 19 adults and the severity of the hæmorrhage may be gauged from the methods employed for its control:

(*a*) Controlled by simple methods *e.g.*, pressure applied to the tonsillar beds, cold pack round the neck, application of hæmostatics, like hydrogen peroxide, Whitehead's varnish, Coagulen Ciba and injection of serum—14 cases.

(*b*) Ligature of bleeding points—4 cases.

(*c*) Stitching of the pillars—4 cases.

(*d*) Ligature of external carotid artery—2 cases.

Except in cases which give a definite history of hæmophilia, in otherwise healthy persons, it is difficult to make a preoperative judgment as to which of them would have postoperative hæmorrhage. Even in those cases where the bleeding time and the clotting time were normal, secondary hæmorrhage occurred. The operation should not be concluded unless one is quite certain that all bleeding has stopped. It might add a few minutes more to the operation, but will save many more afterwards. A shining film of serum forms over the tonsillar beds after tonsillectomy, and its absence may be a warning that oozing may occur. Further, the presence of a white covering of slough in the tonsillar fossæ is always a reassuring sign, and is usually absent, or gets removed in patients who get secondary hæmorrhage. In a recent case of a young man who has had a full course of M. & B. 693, I encountered recurrent hæmorrhage at first from one and then from the other tonsil. The bleeding was venous oozing, and could at length be brought under control by suturing the pillars, injections of Coagulen Ciba, hæmostatic serum, intravenous calcium and vitamin K. I would venture to make a tentative suggestion that the ingestion of M. & B. 693 might have been responsible for lowering the coagulability of the blood.

Pulmonary Complications

Bronchitis—A mild attack of bronchitis is sometimes observed. The usual rise of temperature upto about 99-100°F. is exceeded to about 102°F. with cough and the usual signs in the lungs. The only case of bronchitis in our series was a boy aged about 6 years who had a rise of temperature on the second day after tonsillectomy which went upto 102°F. He was cured in a short time. Nesbit (1934) reviewing 2,357 cases also records one case of acute bronchitis.

Pulmonary tuberculosis—Tonsillectomy *per se* will not give rise to it, but where such a lesion, quiescent or active, already exists there is great danger of its exacerbation. In our series two such cases were encountered, both in young girls. One of them a girl of 15 years had a severe hæmoptysis to begin with and subsequently died, the other one aged 18 years had a prolonged rise of temperature in the afternoon. A skiagram of the lungs revealed an active lesion in the left lung. After proper treatment it healed up and she is now quite well, seven years after the operation.

Lung Abscess—There is a great deal of controversy regarding the incidence of this complication. Some authorities consider that the incidence of lung abscess after tonsillectomy is very high (about 60 per cent), while others, for example, Keen (1931) did not come across even one case out of a series of 9,344 operations which he reviewed. Ormerod (1941) mentions that some 20 per cent cases of lung abscess have had tonsillectomies. Richardson was the first to draw attention to the relationship of tonsillectomy and lung abscess. Fischer and Cohen (1921) reports 5 cases. In our series of cases only one pulmonary abscess was met with. Tirumal Rao, quoted by Mahadevan (1942), has had only one suspected case of lung abscess in about 3,000 tonsillectomies.

The following case of lung abscess as a complication after tonsillectomy is reported:

M. L., 20 years, operation under local anæsthesia, upright position; remained well for 10 days, then had fever with chill. On clinical and radiological examinations a lung abscess was diagnosed. He was cured after sulphonamide and postural therapy.

Moore (1922) has given a comprehensive account of lung abscess following tonsillectomy. His conclusions are that the incidence is higher in recumbent than in the upright position, much more under general

anæsthetic than under local. Debris in the tonsillar crypts was present in a large number of cases so affected, and it was not removed before the operation. He found that the right lower lobe was affected in 40 per cent of the cases, and presence of some other lesion favoured its occurrence. In his estimate, the incidence is not so high as mentioned by some authors, and that only 1 case in about 2,500 or 3,000 is so complicated. As precautionary measures he suggested that tonsils should be cleared of any debris before operation, a local anæsthetic and an upright position during operation should also be adopted as far as possible. A suction apparatus to remove any blood is very helpful, and it is emphasized that the 'bechic reflex' should not be abolished, because in the words of Chevalier Jackson, " 'Bechic reflex' is the watch dog of the lungs. Don't drug it unnecessarily."

Pleurisy—We have had no case of this complication, although Nesbitt (1934) mentions two cases with recovery.

Otitis Media

A pre-existing otitis media which may be quiescent is very likely to flare up. Middle ear inflammation may be set up in an ear which was hitherto healthy, but this is not very common since the defensive mechanisms are quite effective.

In our series two cases have been noted:

Case 1—Child, aged 8 years, pain in the right ear on the second day of operation There was an inflammation of the tympanic membrane. It did not go on to suppuration.

Case 2—A young man aged 22 years, tonsillectomy under local anæsthetic, pain in the ear with inflammatory signs, subsided quickly.

Nesbitt (1934) found that the incidence of earache following tonsillectomy was 2·8 per cent and actual suppurative otitis media occurred in 0·1 per cent only. Amongst hospital patients it was slightly higher, being 1·5 per cent.

High Pyrexia and Septicaemia

Apart from pulmonary complications, high pyrexia is usually the result of infection of the wound with organisms *e.g.*, streptococcus or pneumococcus, or due to septicæmia. Morgan (1934) mentions 11 cases of severe illness due to high pyrexia and septicæmia of which 7 were fatal. This, however, is a fairly large number, specially the number of fatal cases, which evidently is due to the sulphonamide drugs not being in use then. A tansient bacteriæmia, however, is found in a fairly large number of cases soon after tonsillectomy or dental extraction. Fischer and Gottendenker (1938) carried out investigations to this effect and found that in 52 per cent of cases bacteria would be grown from the blood soon after tonsillectomy. However, Southworth and Flake (1938) have found it in only 18·2 per cent. This bacteriæmia does not result in any severe illness and disappears in about 12 hours.

We had four cases of high pyrexia which yielded to sulphonamide drugs:

Case 1—F., aged 19 years, high fever 104°F. on the second day after operation. Blood culture was sterile; throat swab showed pus cells, pnumococcus and staphylococcus; there was no T. B. in the sputum but on skiagram of the lungs the right apex was found to be comparatively hazy. The temperature settled down to normal in 7 days after the administration of sulphonamide.

Case 2—Y. P. S., male, 25 years, tonsillectomy under local anæsthesia, had high fever upto 103°F. for about a week. The fever settled after intravenous injection of 5 c.c. of a 3 per cent solution of sodium salicylate.

Case 3—S. C., boy of 8 years, temperature shot upto 104°F. on the second day after tonsillectomy, subsided in about 4 days.

Case 4—H., male, 13 years, high fever with rigor 2 days after tonsillectomy rising upto 105°F. Administration of Urea-sulphazide failed to bring down fever but M. & B. 693 succeeded and the boy was cured in about 2 days' time.

Paresis of the Soft Palate

This is not an uncommon observation after tonsillectomy, due to injury and inflammation of the soft palate and its movement is much interfered with. The patient complains of regurgitation of fluids through the nose and rhinolalia. Usually it is of a transitory nature disappearing in about a week's time, although Thompson (1936) quotes a case in which it persisted for 24 years. He thinks it was probably functional.

Atrophy of the Uvula

This is rather rare. The one case seen is reported below:

P., male, 20 years; tonsillectomy under local anæsthesia; œdema of the uvula noticed on the second day which gradually shrank to the size of small nodule. There was no injury during operation, and the uvula was quite intact after the operation.

St. Clair Thompson (1937) mentions 2 or 3 cases which he and Negus have seen and have heard of some more.

Change in the Voice

This is of some practical importance, specially in persons whose livelihood depends on the use of the voice. The question is frequently asked whether tonsillectomy would cause any interference with the

voice, specially in singers. It is believed that tonsillectomy may cause some weakness of the voice for some time, say six months, but after this if the operation is done with care causing no laceration of the pillars, and if they are prevented from falling together and causing 'tight palate', there should be no interference with singing. As a matter of fact in those cases where the patient has been getting recurrent attacks of tonsillitis it may actually improve. I know of one patient whose voice became squeaky after tonsillectomy but he had an attack of acute laryngitis after the operation which must be responsible for the change in voice rather than the operation itself.

Deep Cervical Phlegmon

The following is a case report illustrating deep cervical phlegmon:

M.D, female, aged 16 years, tonsillectomy under local anæsthesia, swelling appeared on the second day after the operation beneath the angle of the jaw on the right side. It was red, hot and tender; sulphonamide was given and the inflammation became localized and the abscess discharged through the tonsillar bed.

In this case the cause for the development of the cervical phlegmon was easily found out. While injecting novocaine solution at the lower pillar of the tonsil with a curved tonsil injection needle its curve was directed outwards, and I could feel it suddenly going into a non-resistant area possibly the connective tissue space between the superior constrictor muscle and the buccopharyngeal fascia. Since then I have been careful to see that the needle is never directed outwards while injecting the peritonsillar space at either pole, specially the lower. Shapiro (1930) mentions no fewer than 30 cases of deep cervical phlegmon. He reviews 80 other cases, there were 3 fatalities. In 94 per cent of the cases local anæsthesia was used. Two clinical types are described. (1) deep phlegmon type characterised by trismus, fever and swelling of the neck; (2) a vascular type which manifests itself as septicæmia, thrombosis or embolism.

As regards treatment it is advised that an attempt should be made, to localize the infection and then draining it through the tonsillar fossa as far as possible.

Surgical Emphysema

This is also a rare complication. I have met with it in one case only when I performed tonsillectomy with the guillotine.

S., male, 20 years, tonsillectomy under general anæsthesia by the guillotine method. A few minutes afterwards a swelling appeared on the right side of the neck with typical crepitus of subcutaneous emphysema. A firm bandage over a pad was applied. The air was absorbed in a day.

Rosenbaum (1924) in reporting the case of a woman aged 35, writes that tonsillectomy was done under general anæsthesia when 10 minutes after the operation emphysema developed over the chest, eyelids, face and neck—all disappearing in 3 days. Howie(1936)mentions another case in a woman of 35 years. He believes that it is due to a tear in the pharyngeal aponeurosis, and describes the mechanism of its production as follows: "It is well-known that the swallowing of mucus and saliva after tonsillectomy is a laborious and exaggerated process. The mouth is tightly closed, the tongue pressed firmly against the palate, and a convulsive act of deglutition carried through with a great increase in the intrapharyngeal air pressure. In this case the repeated act of swallowing led to air being forced through to defect in the pharyngeal aponeurosis and its tracking in the directions indicated."

Acute Rheumatism

Girl, 10 years, tonsillectomy under general anæsthesia; posttonsillectomy fever which normally subsides in 2 or 3 days persisted for a month, when myocarditis was discovered and the patient died after about 2 months.

This incident made me think whether tonsillectomy is capable of flaring up a latent rheumatic myocarditis which may be fatal. Stroud *ed al* (1933) thinks that routine removal of the tonsils has reduced the incidence of acute rheumatism, and it is possible that the patient may be made worse than before the operation. Johnson (1930) mentions of a boy who developed acute rheumatism after operation.

Cavernous Sinus Thrombosis

We have had no case of this grave complication. Campbell (1932) records a case of fatal cavernous sinus thrombosis following tonsillectomy by the dissection method under local anæsthetic. Probably it was the result of the vascular type of deep cervical phlegmon, alluded to above which led to a thrombosis of the cavernous sinus.

Brain Abscess

Johnson (1930) describes a case in a boy of 7 years. Tonsillectomy was followed 5 weeks later by acute rheumatism and valvular stenosis. There was a sharp rise of temperature on the fifth day, pulmonary embolism, bronchopneumonia and later on development

of brain abscess. We have met with no such complications in our series.

Glottic Spasm

There has been no case of glottic spasm in the series studied but this complication occurs at times. Keen (1932) describes 5 cases in which it happened and in one case laryngotomy had to be performed.

Diphtheria and Acute Anterior Polyomyelitis

Diphtheria and acute anterior polyomyelitis, specially of the bulbar type, have been observed to follow tonsillectomy. It is, however, on account of the greater vulnerability of the pharynx to these infections, that such complications may arise.

Habitual Dislocation of the Jaw

I have recently observed this complication in a young woman whose tonsils I removed under chloroform anæsthesia. When I saw her about six months after the operation she complained that the lower jaw gets dislocated very easily. In anæsthetized patients where a mouth gag is used it not uncommonly happens that the lower jaw is dislocated forwards temporarily, but this case is unique in that it has persisted. We have to assume that the temporomandibular ligaments suffered rupture which could not heal up normally.

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ON THE ÆTIOLOGY OF ACUTE RHEUMATIC FEVER

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Within the series of rheumatic disorders, acute rheumatic fever rightly claims a particular position. Quite contrary to the signs and symptoms of *chronic* diseases of the joints, so often vague and indistinct, so that sometimes distinction even between inflammatory and degenerative processes appears impossible, acute rheumatic fever presents a rather well determinable clinical condition. The acute feverish beginning, the typical, most dolorous swellings of joints, the characteristic complications and sequelæ, result altogether in a well known and well defined syndrome.

However, that compact unity of clinical aspects fades away as soon as the question of ætiology arises. In the following study, I made an attempt to replace that unity, for ætiological reasons, by two separate conditions, different from each other by anatomical and clinical signs.

Streptococcus or Ultravisible Virus?

Streptococcus hæmolyticus is usually considered to be the organism responsible for acute, rheumatic fever. In fact, for more than thirty years, quite a number of observers succeeded in culturally demonstrating streptococci from various tissues: from periodontium and the teeth, from tonsils, joints, cervical and mediastinal glands, and last but not the least, from endocardium and heart valves.

It is not possible to refer, in a short paper, to all the publications about streptococcus and acute rheumatic fever. A survey of the outstanding literature, throughout the world, has been given in the monograph "Streptokokken infektionen" by von Lingelsheim (1928) in "Handbouch der pathogenen Mikroorganismen" by Kolle, Kraus and Uhlenhuth. Worth mentioning among recent publications is a study by Collis (1939), results of which read as follows: in 17 rheumatic fever autopsies Collis observed growth of hæmolytic streptococcus, in 14 out of 15 tonsils, in 13 out of 27 cervical or mediastinal glands and in 22 out of 42 heart valves.

It has been objected to the theory of streptococcal origin that streptococci in the tonsils present a very common finding, even under normal conditions, that

streptococci in the joints, in spite of the enormous material studied already, are of extreme rarity, and that in all other organs in question growth of streptococcus occurs only occasionally.

The opponents of the theory of streptococcal origin believe that the disease is caused by a specific, ultravisible virus which has not been discovered so far, an opinion founded on the missing of streptococcus in the majority of observed cases, and confirmed by a number of signs and symptoms differing from those originated by streptococcus.

Both opinions being supported by good arguments, the conclusion is permitted that both sides are right: that streptococcus as well as another virus, unknown hitherto, have to be taken into account as being the ætiological factors of acute rheumatic polyarthritis.

It cannot be pretended, of course, that two different bacteriological causes are effecting identical diseases. However, it can be demonstrated that both causes produce a separate infectious disease each, in other words, that genuine, acute rheumatic fever and streptococcal arthritis present two different although somewhat similar diseases, discernible from each other in their pathological and clinical aspects.

Anatomy

Differentiation between genuine rheumatic fever and streptococcal arthritis is possible on histological lines due to the presence or absence of Aschoff's nodules in the connective tissue.

These nodules, presenting a typical granuloma, are understood to be the product of a strictly specific reaction of the tissue to the rheumatic attack. They have been compared with the specific products of two other inflammatory conditions: the syphilitic gumma and the tubercle. The discovery of these nodules, observed already in tonsils, joints, fasciæ, endo- and myocardium, constitutes the main progress of rheumatism research of the last two decades. They present the criterion of acute rheumatic fever, and it was logical that one of the outstanding pathologists suggested to speak of "granulomatosis rheumatica" in lieu of genuine acute rheumatic fever. Aschoff's nodules, as a rule, are present in all those cases, where clinical and bacteriological signs speak against a streptococcal disease. They are missing whenever streptococcal ætiology is evident, a fact of utmost importance, permitting, from the anatomical point of view, certain separation between the two conditions.

It is an item of highest interest for the practitioner that Aschoff's nodule are never observed in the perio-dontium but have been found usually in peritonsillar tissue. On the other hand oral septic processes and foci in periodontal tissue, as a rule, contain streptococci.

The conclusion, therefore, is that genuine acute rheumatic fever is never propagated from the teeth, but quite commonly from the tonsils! Arthritis, propagated from the teeth, is of streptococcal origin.

Clinical Aspects

The clinical manifestations of uncomplicated acute rheumatic fever as well as of uncomplicated streptococcal arthritis are very similar to each other. It is true, as some authors point out, that typical streptococcal arthritis is usually confined to one single or only a few joints, and that occurrence of typical polyarthritis is a somewhat rare event. However, such differences are uncertain.

In attempting to find out differences in the clinical aspects, one has to look away from the more simple cases, and to concentrate on the accompanying diseases and complications.

Diseases of the Heart—The most common complication of genuine acute rheumatic fever is the acute inflammation of the endocardium, an event occurring, unfortunately, in about 60 per cent of all cases—endocarditis simplex, verrucosa. Anatomically, its characteristics are papillar excrescences of the valves with gradual growth of granulation tissue and superficial necrosis. No microorganisms are found.

The other kind of endocarditis that occurs following streptococcal arthritis, endocarditis ulcerosa, is distinguised from the former by suppuration and ulceration of the valves. It is the malignant species of the disease, although by no means essentially fatal. Bacteriologically the main feature of the disease is the enormous number of streptococci, especially in the detritus of valves. Clinically it may be difficult, at times, to differentiate endocarditis ulcerosa from verrucosa, although the former, as a rule, will show more severe symptoms. It is of value to remember, for differential diagnosis, that endocarditis ulcerosa, in contrast to endocarditis verrucosa, is usually accompanied by other signs of septic condition, such as tumor of the spleen, streptococcal nephritis, etc.

Cerebral Diseases—It is well known that acute rheumatic fever is closely connected with that particular disease of corpus striatum, which owing to the remarkable sign of involuntary and incoordinated movements, is called "chorea", dancing disease, (St. Vitus's dance).

The main item of the disease is an inflammatory condition of the putamen and nucleus caudatus. No visible microorganisms have ever been observed in any part of the cerebrum.

As mentioned above, chorea is a typical sequel to acute rheumatic fever occurring solely in connection with the latter.

Streptococcal arthritis, on the other hand, is also followed, occasionally, by cerebral complications, but of quite a different and, unfortunately, most alarming kind, the most common sequel being purulent meningitis.

Eye Diseases—The specific complication of genuine rheumatic fever is rheumatic iritis with its typical signs: discoloration of the iris, irregular pupil, pericorneal injection.

A complication of streptococcal arthritis is streptococcal emboli into the eye, resulting in iridochorioiditis suppurativa or even purulent panophthalmitis, usually leading to complete loss of the eyeball within a couple of days.

Acute Arthritis and Drugs—For practical purposes, by far the most important criterion between genuine acute rheumatic fever and acute streptococcal arthritis, is the different response of both diseases to certain drugs.

The specific remedy of genuine rheumatic fever is, for more than 60 years now, sodium salt of salicylic acid. The effect of this drug is so convincing, so striking that it must duly be counted among our classical pharmaceutics. If in a supposed case of rheumatic fever salicylic acid fails, one should be justified in considering an error of diagnosis. Usually in such an event, there will be a case of streptococcic arthritis, and treatment with sulfanilamide will then be indicated.

It is noteworthy that sulfanilamide does not influence genuine acute rheumatic fever in the least, according to reliable observations by Massell (1937), Swift *et al* (1938).

On the other hand, it has been pointed out by Gantenberg and Thimme (1935) that sulfanilamide should be of value in making a differential diagnosis "exjuvantibus" between arthritis of infective origin and specific rheumatic fever, since the latter does not respond to the drug.

Summary

Concluding I must admit, with reference to the annexed table that, as a matter of fact, not all clinical cases permit such a clear distinction between the two groups as may be expected in theory.

	Specific acute rheumatic fever	*Streptococcal arthritis*
Histology	Aschoff's nodules present in connective tissue.	No Aschoff's nodules.
Clinical Aspects	Cardiac complication: endocarditis verrucosa, (streptococci in endocardium negative).	Cardiac complication: endocarditis ulcerosa, (streptococci in endocardium positive).
	Cerebral complication: chorea minor.	Cerebral complication: purulent meningitis.
	Ophthalmic complication: iritis rheumatica.	Ophthalmic complication: purulent iritis, panophthalmitis.
Pharmacology	Responds to salicylic acid.	Responds to sulfanilamide.

If one remembers that the most probable entrance focus of the unknown virus of acute rheumatic fever must be looked for in the tonsils—as proved by the occurrence of Aschoff's nodules in the peritonsillar tissue—one will easily realise that an accompanying infection with streptococcus is not only possible, but even most probable.

However, such considerations should not prevent us from separating the two conditions from each other. May I quote, in this connection, another similar coincidence in human pathology: The incidental occurrence of a "chancre mixte", containing spirochæta pallida as well as Unna-Ducrey's bacillus, would never permit us to mix up the pathological and clinical pictures of the two conditions, syphilitic ulcer and soft sore.

The most striking distinction between genuine acute rheumatic fever and streptococcal arthritis is: confrontation of the respective germs is not possible so far, as the virus of acute rheumatic fever is unknown to us for the time being. So any bacteriological argumentation, which is restricted to speak of positive and negative findings of streptococcus only, without due consideration of the hypothetic germ of acute rheumatic fever, remains to be rather unsatisfactory.

Therefore, distinction between the two diseases has to be made according to lines, other than bacteriological, a task which has been attempted herewith.

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Special Article

THE TRIDOSA THEORY

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In this communication an attempt has been made to establish the basis of the *Tridosa* theory which, again, in its turn, is the basis of the Ayurveda. The method adopted in this study is to collect as much material as possible from the *Samhitas* relating to the functions, normal and abnormal, of *Vayu, Pitta,* and *Kapha. Carak Samhita* and *Susruta Samhita* have rather exclusively been used for the study, only because these alone represent the conception of *Tridosa* as described by the originators of this theory. The subsequent writers have only summarised the original works adding their own interpretation so they have not been taken into consideration excepting casually. This material which is fairly rich has been scrutinised in order to find whether it can be classified and grouped in any standard and scientific way. The result is remarkable. All this material can be adequately grouped under headings which can explain all the symptoms and functions grouped under each and at the same time agree essentially with the modern physiological doctrine. The *Tridosa* theory is thus found to be truly scientific and based on exact science. It is remarkable that good many things visualised by the *Rishis* of remote ages have been proved on experimental basis and that only very recently.

The order of study under each head *Vayu, Pitta* and *Kapha* has been as follows:—

(1) Material collected from the *Samhitas.*
(2) Grouping of the above material under appropriate heads.
(3) Arranging these groups according to 5 kinds described in the *Samhitas.*
(4) Seat of the *Dosa.*
(5) General functions, normal and abnormal, explained.
(6) Properties of the *Dosa* and the temperament due to preponderence of the *Dosa.*
(7) Causes of derangement.

दोष धातुमलमूलं हि शरीरम् (S. S. 15|3)*

The material body is composed of (1) constituents divided into 7 categories, called *Dhatu,* (2) the waste products, called *Mala,* (3) and the controlling authority of the *Dhatu* and *Mala* are the *Tridosa, Vayu, Pitta* and *Kapha* of which *Vayu* is the life, *Pitta* the fire, *Kapha* the fluids of the body as mentioned by Susruta, **तत्रवायुरात्मै धात्मा, पित्तमाग्नेय, श्लेष्मा सौम्य इति** (S. S. 42).

The three fundamental functions of the body—(1) movements **(सञ्चालन)** (2) heat production **(स्वेदन)** and (3) lubrication **(स्नेहन)** are being carried out respectively by *Vayu, Pitta* and *Kapha.*

The function of movement is being carried on by nerve impulses acting mainly through the muscles both voluntary and involuntary. The function of heat production is carried on by disintegration and metabolism of the food materials inside the body. The function of lubrication is carried on by the property of supporting each individual cells so as to allow them to perform conjoint action. These three *dosas* thus influence on the 7 groups of tissues elements called *Dhatus* which constitute the human body and also the excreta *Mala* to perform day to day function of the body.

Separate communications, under publication, have been devoted to the study of these three functions of the body in all their aspects. It has also been pointed out that when deranged, *Vayu, Pitta* and *Kapha* become evident respectively by pain **(शूल)** burning sensation **(दाह)** and swelling **(शोथ)** as the chief manifestations.

It is sought to explain *Vayu* as the nervous impulse conducted through the nerves, in which *Vayu* represents the electricity and the nerves the dead wires which only act as the vehicle. So the nerves themselves are not *Vayu,* it is the nerve impulse which is *Vayu.* A new building may be fitted with the proper wiring system connected to the electric fans, bulbs, radio receiver, refrigerator, etc., but none of these will do the function until the electric supply corporation connect the same with their mains. The building without electric supply but with inside wiring complete may be compared with a dead body with all the nerves intact.

As an electric machine with adequate wiring is inactive without electricity so also the animal body with all nerves intact but deprived of *Vayu* is dead. *Vayu,* like electricity is thus invisible and revealed only by its effect, *i.e.,* **अव्यक्तव्य कर्म्मांच।**

It is sought to explain *Pitta* as the function of all glands of our body, particularly the digestive glands which digest the food making it available for the energy and heat, *i.e., Agni* of our body. *Kapha* represents the body water and the supporting and lubricating systems of our body. This can now be taken up separately.

VAYU

According to the order of study stated above, in the first place the materials from the *Samhitas* have been collected, a scrutiny of which presents us with 313 symptoms as manifested by normal and abnormal *Vayu.* These are as follows:—

(1) The 80 symptoms of abnormal *Vayu* (C. S. 20|9) =80 symptoms.
(2) Symptoms of abnormal *Vayu* according to site of affection (C. C. 8|22-34)=72 symptoms.
(3) Symptoms due to 5 different kinds of *Vayu* (C. C. 28|4)=16 symptoms.
(4) Symptoms of one *Vayu* covered by the other (C. C. 28|67, 68)=41 symptoms.

* In the references from the *Samhitas* the following abbreviations are used. The first letter, S or C indicates Susruta Samhita and Caraka Samhita respectively. The second letter S, C or N indicates Sutrasthanam, Cikitshasthanam and Nidanasthanam respectively. The first figure indicates the chapter and the last figure the number of the paragraph.

(5) Symptoms of *Vayu* affection according to site (S. N. 1|21-23)=36 symptoms.
(6) Symptoms of abnormal *Vayu* (C. C. 28|8)=21 symptoms.
(7) Symptoms of abnormal *Vayu* (Astanga-S. 21|46-47) =24 symptoms.
(8) Symptoms of abnormal *Vayu* (C.-S. 20|10)=23 symptoms.

This makes a total of 313 symptoms due to normal and abnormal *Vayu* as collected from *Samhitas.*

The next order of the present study has been to group the above material under appropriate heads. Following this order it has been found that all these 313 symptoms can be very appropriately classified under 9 groups as follows:—(1) Symptoms due to normal and abnormal mental functions, (2) those of special sense organs, (3) those of circulation, respiration, deglutition, (4) of excretion, (5) of digestion, (6) due to vasomotor system, (7) those of sensation, (8) those of motor function, (9) speech.

The third order of the study has been to arrange those groups according to 5 kinds of *Vayu* as described in the *Samhitas.* The attempt has been very successful. The symptoms classified under the first 3 groups (mental, special senses, those of circulation, respiration and deglutition) exactly fit in with manifestation of *Pran Vayu* as mentioned in the *Samhitas.* Similarly the group 5, *i.e.,* those of digestion are the function of *Saman Vayu,* the group 4, *i.e.,* those of excretion as of *Apan Vayu* while the groups 6, 7, and 8, *i.e.,* vasomotor symptoms, sensory and motor symptoms as of *Vyan Vayu* and finally the group 9, *i.e.,* speech as of *Udan Vayu.*

An exhaustive study of what has just been said as also the other aspects of the order of study, *i.e.,* (4) seat of *Vayu,* (5) explanations of general functions, (6) properties of *Vayu* and the symptoms of *Vayu* temperament and lastly (7) causes of derangement of *Vayu* form the subject of another communication to be published shortly in this journal. Here we discuss *Vayu* in a general way.

Let us discuss the function of each of the 5 *Vayus* separately. For the sake of convenience we take these in the order of *Vyan, Udan, Saman, Apan,* and *Pran* instead of the conventional arrangemet.

Regarding the function of *Vyan Vayu* we find from C. C. 28|7 and S. N. 1|17 that *Vyan Vayu* residing all over the body produces movements, flexion, extension, closing and opening of the eyelids, regulate flow of sweat and blood, circulation of blood, lymph and chyle throughout the body. The function of carrying sensation from every part of the body although not mentioned anywhere but from the 80 symptoms of abnormal *Vayu* (C. S. 20|9) nearly 30 are due to abnormal sensations. The function of sensation I have atributed to the function of *Vyan Vayu.*

Vayu is divided into two categories according to the functions, whether external or domestic. All external functions are those which are under the control of will while the internal functions are those beyond the jurisdiction of the will. The functions maintained by will are again divided into two sections those regulated by the central nervous system, *i.e.,* the brain and spinal cord and other by the peripheral nervous system—the sensory and motor nerves. The peripheral nervous system is undoubtedly the functionating agents, *i.e.,* the magistrates and the police in any government while the central nervous system represents the governor and the ministers-in-charge. This simile can be extended still further. *Vyan Vayu* being present all over the body, performs (1) the sensory nervous functions, *i.e.,* the minister-in-charge of information to the government and (2) the motor nervous functions, *i.e.,* minister of interior and (3) the vasomotor functions, *i.e.,* of the transport minister. Autonomy has been given to the vasomotor system of the body which regulates the blood flow to every part of the body by the contraction or dilatation of the capillaries thus making their lumen narrower or bigger. This is autonomous because it is not controlled by will. *Vyan Vayu* represents the sensory, motor and vasomotor systems of the body and according to the simile, thus performs the functions of minister-in-charge of information, minister of interior and transport minister.

Udan Vayu, situated at the throat, has got only one function and that is that of the propaganda minister by speech (C. C. 28|5). Although this is a single function but the mechanism is complicated. If we take the radio broadcasting station as only one of the means of its function where there is only sound being utilised we can have some idea of the effect of sound or speech. This not only serves the purpose of broadcasting the announcements as is with all animals, the herd is thereby informed of imminent danger or serves the purpose of expressing dissatisfaction by cry, pleasure by loud laughter or pain by cry. But with human beings, through the action of speech, the teachers throughout the ages have taught the arts and science in all their branches, *i.e.,* the function of education minister as well. The more developed and trained *Udan Vayu* the individual becomes better politician and statesman. With jugglary of words an individual can speak for hours, the audiance admiring the speech thinking that the problem in question has been dealt with threadbare and the speaker has explained his personal opinion but the real fact might be just the reverse. *Vyan Vayu* and *Udan Vayu* are directly controlled by will, the central government *i.e.,* the cerebrospinal system and they are worked through the peripheral nervous system.

Saman Vayu and *Apan Vayu* on the other hand, purely belong to the departments enjoying complete autonomy, as they are not under the control of Will. These are entirely domestic affairs of the government. *Saman Vayu* is absolutely beyond the control of Will while although autonomic *Apan Vayu* requires the help of *Vyan Vayu* to complete its function. The minister-in-charge of agriculture, industries and finance in our body government represented by the *Saman Vayu* has been given autonomic powers, outside the jurisdiction of the governor, the Will. How this is done is now explained. According to Caraka (C. C. 28|6) and Susruta (S. N. 1|16) *Saman Vayu,* remaining in the stomach and intestines regulates digestion by vascular and secretary activity of the stomach and small intestine. I have ascribed *Saman Vayu* as the solar plexus, semilunar and superior mesenteric ganglia of our autonomic system. After the food has reached the stomach the whole process of digestion, is done by the *Pachaka Pitta* i.e., the product from the industrial centres or factories—the digestive glands, being regulated by *Saman Vayu* the abdominal

sympathetic system. *Saman Vayu* thus converts the food into the final production of nutrient material, its separation from the waste materials, as performed under the jurisdiction of the departments of agriculture and industries and finally their absorption into the body as supplier of energy and life as *Rasa Dhatu* the finance of our body government which fill up the coffer of the finance minister. These are all done by *Saman Vayu* and that without our consciousness. This goes on during sleep, i.e., when the governor, the Will, is inactive, and also when the individual is under anæsthesia or when unconscious due to disease i.e., when the governor is absent. *Saman Vayu* thus acts as a dictator having the whole charge of agriculture, industry and finance.

Apan Vayu, the minister-in-charge of sanitation, whose chief function is to maintain public health by elimination of the waste products, and that in our body mainly in the form of fæces, urine and fœtus, not constantly, but at regulated intervals is also autonomic and is beyond the control of Will. According to Caraka (C. C. 28|8-9) and Susruta (S. N. 1|19-20) *Apan Vayu* is responsible for the acts of micturition, defecation and parturition for which reason I ascribe *Apan Vayu* as the inferior mesenteric ganglion of our autonomic nervous system. *Apan Vayu* regulates the excretion by unconscious contraction of the sphincters which continues its function during sleep or unconsciousness and only at definite intervals the sphincters are relaxed resulting in the passage of feces, urine or the fetus. So far it is autonomic. Just before the acts of defecation, micturition or parturition, *Apan Vayu* sends the message to the minister of information, which being guided by Will can control these acts for sometime at least but not indefinitely. The act of parturition *i.e.,* the birth of the child, is beyond the control of Will. The minister of information can also guide *Apan Vayu* to perform the function of defecation and micturition prematurely. So far regarding the co-ordination of *Apan Vayu* with the minister of information. The other help of *Vyan Vayu* the minister of interior *i.e.,* the motor functions, are invariably been requisitioned by *Apan Vayu* to finish its function. During defecation, micturition and parturition the contraction of the abdominal and other muscles in the form of bearing down strain become invariably necessary to complete the act. This shows that *Apan Vayu* although autonomic requires help from *Vyan Vayu*—sensory system (minister of information) and motor system (minister of interior).

Now comes the most important *Vayu* the *Pran Vayu.* As the name implies it is the life itself. According to Caraka (C. C. 28|4) and Susruta (S. N. 1|13-14) the functions of *Pran Vayu* in normal condition, are spitting, sneezing, belching, respiration, deglutition etc. From other passages it is seen that *Pran Vayu* also regulates the mental functions as well as the organs of special senses. Caraka locates the site of *Pran Vayu* in the head, thorax, ears, tongue, mouth and nose. I have identified the *Pran Vayu* as the brain, the nerve fibres to the special sense organs of smell, sight, hearing and taste, and the superior and inferior cervical ganglia. *Pran Vayu* thus directs three distinct functions, (1) the mind, (2) special senses, and (3) the most important life saving and life building functions, *i.e.,* circulation and cardiac action (excluding the capillary circulation which is controlled by *Vyan Vayu*), respiration and intake of food. The mind, the originator of Will, director of all important functions, is undoubtedly of vital importance. The special senses (excluding sensation of skin which has already been dealt with under *Vyan Vayu*) are also of vital importance but the functions grouped under the third heading *i.e.,* circulation, respiration and intake of food are most vital. Life can exist without mind during **सुषुप्ति** or during unconsciousness, life can exist without all special senses, but life cannot exist without intake of food, or for long without respiration or even for a moment without circulation. In fact, the cause of every death is either failure of circulation or of respiration. This last named functions of *Pran Vayu* are of 3 kinds according to its relation with the effects of Will. The function of *Pran Vayu* with respect to circulation is absolutely autonomic and beyond the control of Will, except with some *Yogis.* Respiration is ordinarily autonomic and goes on during sleep or unconsciousness but can also be regulated by Will. The intake of food is under the jurisdiction of Will. So far with the ordinary functions of *Vayu* in its 5 forms.

Vayu is said to be **यन्त्रतन्त्रधरः।** What is **यन्त्र** and what is **तन्त्र ? यन्त्र** in ordinary sense means machines. What are the machines of our body? They are the heart, lungs, and all the glands of our body as so many factories with liver the chief and biggest factory. **तन्त्र** may mean the supporting framework *e.g.,* the bones, muscles, blood and the blood channels and the body water which supports and bathes each individual cell, the microscopic elements of our body. We have already mentioned the function of *Vayu* in regulating heart and lungs, the most vital machines of our body. Regarding the other function of *Vayu* as **यन्त्रतन्त्रधरः** we can find from what has just been enumerated as **यन्त्र** and **तन्त्र** that they are nothing but *Pitta, Kapha* and different *Dhatus.*

Pitta represents the glands of our body, some with internal secretion *e.g.,* adrenal, thyroid etc. and others with external secretion *e.g.,* sweat, sebaceous, salivary glands etc. and some with both external and internal secretion *e.g.,* testes, pancreas etc.

Kapha represents the supporting framework, of which body water is most important, others are the 7 *Dhatus, Rasa* (chyle), *Rakta* (blood) etc.

Vayu thus regulates all the functions of all machines and supporting framework of our body and so appropriately called **यन्त्रतन्त्रधरः।** This comes in close meaning when we consider what Sarngdhar says

पित्तः पङ्गुः कफ पङ्गु, पङ्गवोमलधातवो।
वायुना यत्र नोयन्ते तत्र धावन्ति मेघवत्॥

PITTA

The same order of study, as mentioned at the beginning of this communication, has been applied to the study of *Pitta* as well.

(1) From the material collected from the *Samhitas* we find the following: *Pitta* produces digestion or indigestion, vision or loss of vision, normal or abnormal temperature of the body, normal or abnormal colour of the body, courage or cowardice, fear or bravery, anger or the reverse, pleasure or displeasure, **मोह** and **अमोह,** satisfaction or dissatisfaction. (C-S. 12/15).

Again, sight, digestion, heat production, hunger and thirst, softness of the body, radiance, cheerfulness, intelligence are the functions of normal *Pitta*. (C-S. 18/43).

Further, colouration, digestion, vitality, strength, intelligence —these are produced by five kinds of *Pitta* (S. S. 15/3).

Again, that *Pitta* which digests all kinds of food materials, separates them into chyle, urine, feces and supplies thereby the essential fire for the nutrition of the body is *Pachaka Pitta*. The *Ranjak Pitta* imparts red colour to the chyle. The *Sadhak Pitta* serves to fulfil one's desire. *Alochaka Pitta* gives the vision. *Bhrajaka Pitta* gives the glow and complexion of the skin. (C. S. 20/9). The symptoms due to abnormal *Pitta* are 40 in number. (C. S. 20/16). These are: (1) sensation as presence of fire by the side. (2) sensation as in burn, (3) burning sensation all over body, (4) as in severe burning all over body, (5) as if smoke is coming out of body, (6) acid eructation, (7) burning from acidity, (8) internal burning sensation, (9) in some local area, (10) rise of temperature, (11) excessive perspiration, (12) local perspiration, (13) bad odour of body, (14) cracking of local areas. (15) suppurative blood, (16) suppurative muscle, (17) burning sensation of skin. (18) burning sensation of muscle, (19) cracking of skin and muscle, (20) cracking of inner layers of skin, (21) red patches, (22) hæmorrhage, (23) jaundice (green), (24) jaundice (yellow), (25) black spots, (26) cracking of muscle and skin of armpit, (27) jaundice, (28) bitter taste in mouth, (29) bad odour in mouth, (30) excessive thirst, (31) non-satiety after meal, (32-36) suppuration of mouth, throat, eyes, rectum and penis, (37) loss of blood or loss of vitality, (38) loss of vision, (39) green colouration of eyes, urine and stool, (40) yellowish colouration of eyes, urine and stool.

(2) In attempting to classify the above material under appropriate heads we find that all of them can be adequately grouped under the following 6 groups.

(*a*) Symptoms due to burning sensation, rise of temperature, perspiration; (*b*) Symptoms due to secretion of sebum, infective and suppurative processes of the skin; (*c*) Symptoms due to disturbance of digestion; (*d*) Pigmentation, hæmorrhage and jaundice; (e) courage, fear etc.; (f) Vision.

(3) Arranging these groups according to the 5 kinds of *Pitta* as described in the *Samhitas* we find that the groups (*a*) and (*b*) represent the *Bhrajaka Pitta;* group (*c*) *i.e.,* digestion, represents *Pachaka Pitta;* (*d*) pigmentation etc. as *Ranjaka Pitta;* (*e*) courage, fear etc. as *Sadhaka Pitta;* and (*f*) vision as *Alochaka Pitta.*

Now let us scrutinise the physiological basis of the headings in each group with a view to explain the symptoms grouped under each 1. The first group is dealt with under two headings—

(*a*) Body temperature: Human body temperature is the balance or difference between the heat production and heat loss. Heat production takes places in the tissues by the oxidation of the assimilated products of foodstuffs from the blood. Heat loss takes place mainly through the skin by evaporation of sweat and also through the lungs due to pulmonary ventilation. Fever is not caused by increased production of heat but by retention of excessive amount of heat due to the disturbance of heat regulating mechanism of our body. The normally high internal temperature is being constantly carried to the surface by the blood, and coming to the skin the heat is lost by radiation, conduction and/or convection. The more the production of sweat the more the heat loss. Thus we find that heat is produced in the tissue and is lost in the skin by sweating. Let us now consider how *Pitta* performs this function. Our foodstuffs are mainly composed of protein, carbohydrate and fat. Of these the carbohydrate and fat supply the most heat and energy. The ultimate product of digestion of carbohydrate is glucose with a chemical formula of $C_6H_{12}O_6$. After absorption this glucose derived from digestion of carbohydrate circulates through the blood and carried to different parts of the body particularly the liver and muscles. Blood also carries hæmoglobin inside the red blood cells which in its turn carry oxygen from the lungs during respiration. This oxygen also reaches all organs and tissues. The more functionating the organ or tissue *e.g.,* the liver and muscle the more oxidation of the glucose takes place by the action of oxygen carried by the blood. Thus we find one molecule of glucose uniting with 6 molecules of oxygen produces by oxidation, 6 molecules of carbon dioxide and 6 molecules of water and heat and energy. This is indicated by the formula $C_6H_{12}O_6 + 6O_2 = 6CO_2 + 6H_2O$.

	CO_2	H_2O	Calories
It has been estimated that 1 gm. of glucose consumes 1·067 gm. of oxygen & produces ..	1·467	0·600	4·1
1 gm. of fat consumes 2·876 gm. of oxygen & produces ..	2·805	1·071	9·3
1 gm. of protein consumes 1·382 gm. of oxygen & produces ..	1·522	0·395	4·1

As a result of this reaction heat and energy are produced which are calculated in terms of calorie. A calorie is defined as that amount of heat required to raise the temperature of one Kilogram (1000 c.c.) of water by 1°C from 15° to 16°C or that amount of energy required to raise 327 Kilogram of weight to a height of 1 meter. From this definition we find that 1 gm. of glucose (sugar) gives 4·1 calories *i.e.,* by its oxidation it produces that amount of energy which can raise a weight of 1340 Kilogram (approximately 31 munds).

It has been estimated that a normal individual at complete muscular and mental rest (called basal metabolism) requires about 1600-1700 calories per 24 hours or 66 calories per hour. This rate is considerably increased during work. Thus a man sitting still will require 100 calories per hour, with slight work 300 calories and with heavy work 600 calories per hour. An athelet in one hour's foot ball match will require 1700 calories *i.e.,* the basal metabolic rate of a normal individual for 24 hours. All this process of metabolism is carried out by *Pitta* which is thus called *Agni* (fire) in *Ayurveda* and is particularly called *Dhatwagni* or tissue fire. The other *Agnis* are *Kayagni, Bhutagni* and *Pachakagni.* These will be described later on. Heat production and maintenance of body temperature are thus produced by *Pitta.*

(*b*) Sensation of heat, burning sensation and perspiration: These sensations are independant of the actual temperature of

the body and depend on two factors (1) amount of blood in the skin. The more the vessels are dilated the more the feeling of warmth *e.g.,* in emotions, although temperature of the body may be normal. In malaria the vessels of the skin are contracted, so there is a feeling of chill and shivering although the temperature might be 104°F. (2) The temperature at the nerve endings of the skin plays the main role for the burning sensation which again is mainly dependant on the secretion and evaporation of sweat. Increased evaporation of sweat makes the surface colder and so sensation of cold is perceived. This explains why we feel relief when under the fan or a breeze than in stagnent air, the temperature of the air remaining same. *Thus we find that the sweat glands are responsible for all the symptoms included under the first group.*

2. The second group indicates the symptoms due to secretion of sebum, infective and suppurative processes of the body. This, again, depends on another set of glands in the skin, *i.e.,* the sebaceous glands, the secretion of which, called sebum, continually being discharged outside gives the glossy appearance of the skin and protects the skin from infective process. Thus the manifestations included in the first two groups of symptoms satisfy adequately the function of *Bhrajaka Pitta* as described in the *Samhitas.*

3. The third group represents the *Pachaka Pitta* the chief function of which is digestion and assimillation of the food. *Pachaka Pitta* consists of the secretions of the (1) salivary glands, (2) the glands of the stomach, (3) the pancreas, (4) small intestine and (5) the liver. All these secretions perform their function by the presence of enzymes in them having some particular function to digest some proximate principles of our food. Thus the saliva acts through the enzyme ptyalin, splitting the carbohydrate up to the stage of maltose and dextrin. The gastric secretion acts through the enzyme pepsin acting with hydrochloric acid digesting the protein constituent of the food up to the stage of peptone. The pancreatic secretion acts through 3 enzymes (*a*) trypsin, which ferments protein into peptone and further to aminoacids; (*b*) amylase, which like ptyalin of saliva, converts starch into maltose; (*c*) lipase which splits up fat into fatty acids and glycerine. The secretion from liver, the bile, helps the action of lipase in the digestion of fat. As the bile does not contain any enzyme it has got no independent action. The intestinal juice contains powerful enzymes, *e.g.,* (*a*) enterokinase which increases the function of trypsin of the pancreatic juice in digesting proteins into the final product aminoacids; (*b*) invertase, maltase, lactase which break down the carbohydrate elements into the final product glucose.

The changes of the process of digestion is shown in the figure where each ferment is represented by a cutting instrument. (See Fig. 1B, the rectangles to the right of the figure).

4. The fourth group resolves itself into jaundice, hæmorrhage, eruptions, and pigmentation of the skin, the functions of *Ranjaka Pitta.* Modern physiology teaches us that the red blood corpuscles which are being constantly produced in the bone marrow appears red because of the red pigment the hæmoglobin contained in it. The red cell circulates throughout the body for a period of about 3 weeks, the usual span of its life and then destroyed. The hæmoglobin thus set free is converted into bile by the cells of reticulo-endothelial system in spleen and liver and bone marrow, and then excreted by the liver as bile which passes into the intestines during digestion or stored in the gall bladder when not required immediately.

Modern science has established the fact that the red blood cells are produced in the bone marrow. It had been a severe strain on the physiologists, till very recently, to think in terms of Ayurveda that the liver plays a great role in the formation of blood. I quote the following from *Wright's Applied Physiology,* 1940 Edition, Pp. 360, 340, 375.

"It is now established that there is a special hæmatinic (hæmatopoietic) principle which is necessary for the normal formation of blood corpuscles from its precursor the megaloblast present in the bone marrow. Deficiency of this hæmatinic principle is the cause of the macrocytic anæmias *e.g.,* pernicious anæmia, nutritional anæmias etc. This principle is found mainly in the liver. Although the active principle has not yet been isolated, but is known not to be iron, protein, or any of the vitamines.

"From the middle of fœtal life to about one month before birth, the liver and spleen are important sites of blood formation. In times of emergency the liver and spleen may resume this blood-forming function in post-natal life.

At about the middle of fœtal life the bone marrow begins to function as a blood-forming organ and is the sole region where red cells are formed after birth.

"During the later months of fœtal life the spleen is actually engaged in forming red blood cells. The spleen forms an important reservoir of red blood cells which may be called on in times of emergency".

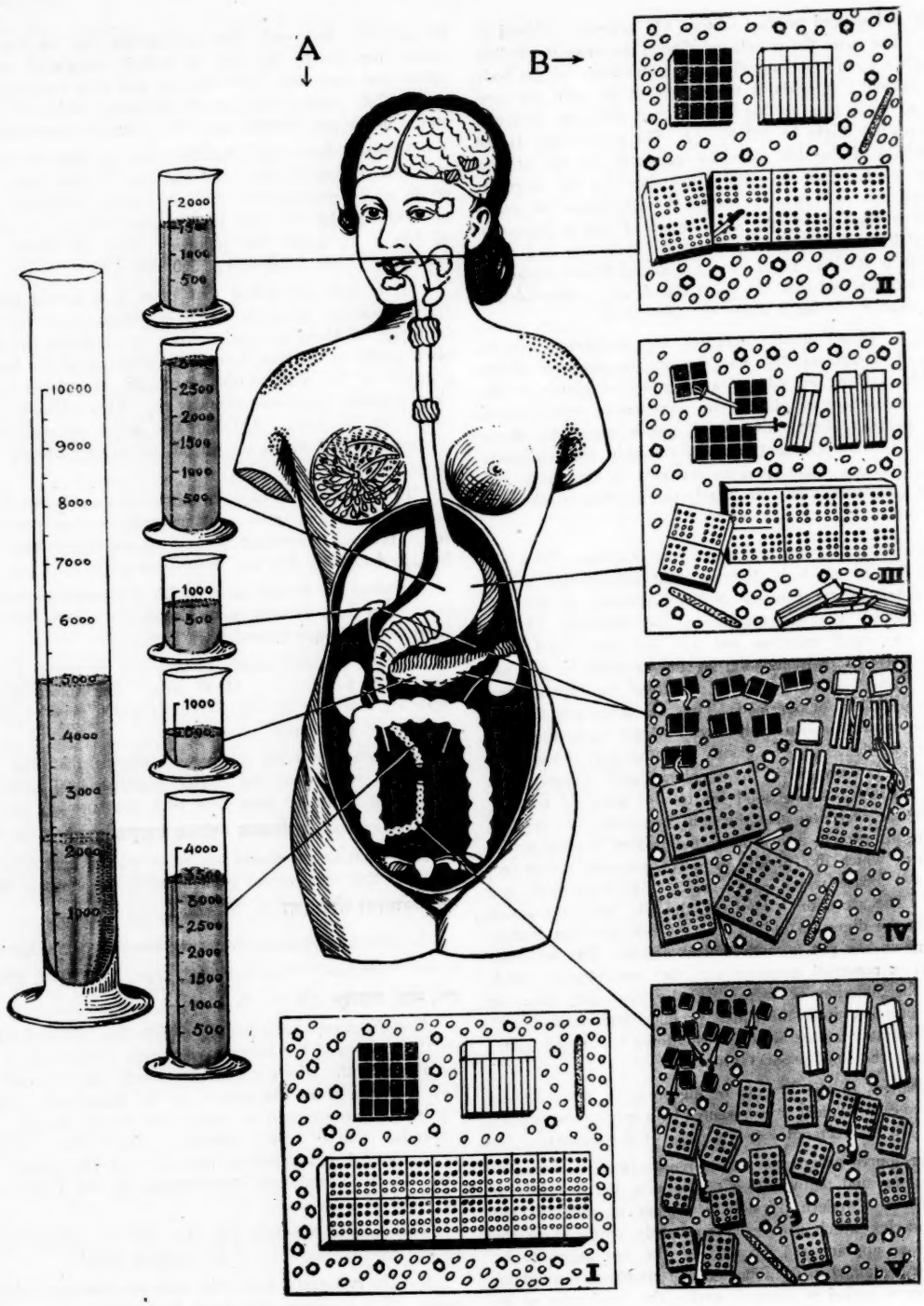
It appears amazing when we compare this very recent knowledge with what the *Rishis* declared 4000 years back. The spleen and liver have thus been appropriately mentioned as the seat of blood **शोनितस्य स्थानम् यकृत्प्लीहानौ** (S. S. 21/16). These are not only the seat but Susruta goes further when he says that they are derived from blood in the embryo **गर्भाशय यकृत्प्लीहानौ शोनितजौ** (S. N. 4/25).

5. The fifth group indicates the function of *Sadhaka Pitta,* in the production of courage, fear, anger etc. **शौर्य्यं, भयं, क्रोधं, हर्षं, मोहं प्रसादम् इति** (C. S. 12/15).

Modern physiologists are of opinion that all these qualities are the function of the secretion of the ductless glands of which the adrenals, the pituitary and the gonads play the chief role. The secretion from the cortex of the suprarenal (adrenal) regulates the production of anger, that of the medulla of the suprarenal, that of fear. Courage is due to the secretion of the suprarenals, the anterior pituitary and the gonads. The effect of the gonads are demonstrated in the eunuchs after castration of the testes.

This proves definitely that the *Sadhaka Pitta* is nothing but the internal secretion of the ductless glands.

6. The last group deals with only one function and that of vision. Here it may appear simple to explain but on scrutiny it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500
I
II
III
IV
V

FIG. 1

appears that what actually represents the *Alochaka Pitta* is not clear. The humours of the eye, I have attributed as *Abalambak Kapha* being fluid in nature. *Alochaka Pitta* might represent the rhodopsin (visual purple) in the retina or the lachrymal gland or both. I think of the lachrymal gland only because all the other varieties of *Pitta* definitely represent secretary glands and if *Alochaka Pitta* is found to be a gland, the identification of *Pitta* in our body becomes absolutely clear.

alimentary tract as *Pachaka Pitta* act as machines to breakdown the food material to be absorbed in readily utilisable forms. These elementary products of food material supply energy to the whole body, liver being the biggest factory for this purpose. The secretions of sweat and sebaceous glands of the skin, the *Bhrajaka Pitta* of our body maintain the constant body temperature and glossy appearance of the body; liver and spleen the seat of *Ranjaka Pitta* regulate the forma-

LEGEND FOR FIGURE 1

FIG. 1. REPRESENTS (A) THE GLANDS OF OUR BODY PRODUCING DIFFERENT 'PITTAS' (SHOWN IN THE HUMAN FIGURE IN THE CENTRE OF THE DIAGRAM) WITH THE QUANTITY OF THEIR SECRETIONS DURING 24 HOURS AND THE TOTAL QUANTITY OF BLOOD (SHOWN TO THE LEFT SIDE OF THE DIAGRAM IN THE FORM OF MEASURE GLASSES) AND ALSO (B) THE DIGESTIVE ACTION OF THE 'PACHAKA PITTA' ON DIFFERENT CONSTITUENTS OF OUR FOODSTUFF (SHOWN IN THE DIAGRAM AS 4 RECTANGLES TO THE RIGHT MARKED II, III, IV, AND V). THE RECTANGLE IN THE CENTRE BELOW REPRESENTS OUR BASIC FOODSTUFF.

The lines drawn from the different glands of the body as shown in the human figure to the measure glasses on the left indicate the quantity of their secretions and to the rectangles to the right their digestive action on the proximate principles of our food.

A. THE GLANDS OF OUR BODY PRODUCING DIFFERENT 'PITTAS' AND THE QUANTITY OF THEIR SECRETIONS—The glands with duct and external secretion are represented in the diagram without any shade while the ductless glands having internal secretion are represented shaded. Those with both external and internal secretions, *e.g.*, pancreas are represented as partly shaded and partly without shade.

Pachaka Pitta—(*a*) Salivary glands, 3 in number on each side of the mouth, are the submaxillary, parotid and sublingual, producing the secretion called saliva amounting to 1500 c.c. in 24 hours. (*b*) Mucous glands of the stomach producing the gastric juice, 3000 c.c. in 24 hours. (*c*) Pancreas producing 500 c.c. of the pancreatic juice. (*d*) Liver producing 750 c.c. of bile. (*e*) Small intestine producing 3500 c.c. of succus entericus.

Bhrajaka Pitta—Sebaceous and sweat glands all over the skin. These are shown in the figure as dots over the shoulders and in the axilla. The female breast is a modified sebaceous gland.

Alochaka Pitta—(?)—Probably lachrymal glands situated at the outer angles of the eye, and the visual purple of the retina.

Sadhaka Pitta—The glands of internal secretion, *e.g.*, the pituitary and pineal inside the skull, thyroid and the parathyroid in the neck, thymus in the chest, pancreas and suprarenal in the abdomen and ovary and testis.

Ranjaka Pitta—The hæmatinic factor in the liver and spleen producing blood. The total quantity of blood in our body, as represented in the measure-glass to the extreme left of the diagram, is about 5000 c.c., of which 45%, *i.e.*, about 2250 c.c. is the volume of corpuscles and the remaining portion, the plasma.

B. THE DIGESTIVE ACTION OF THE 'PACHAKA PITTA' ON DIFFERENT CONSTITUENTS OF FOODSTUFF (SHOWN IN THE 4 RECTANGLES TO THE RIGHT OF THE DIAGRAM MARKED II, III, IV AND V).

In I (the rectangle at the centre below) the basic foodstuff is represented schematically.

Protein is represented in the left upper corner of I by a big black square with 16 compartments. Each compartment, as in V represents the aminoacid particle, the final product of protein digestion. A unit of 4 such compartments is shown to represent the peptone particle as in III, the gastric digestion.

Fat, which is composed of glycerol and 3 fatty acids, oleic, stearic and palmitic, is represented in the upper part of I. This has been shown in IV where the glycerol portion and the three fatty acid portions are separated by the action of liapse aided by bile from liver. In V these have been shown to have joined together ready for assimilation as body fat.

Carbohydrate is shown as a big block, several times the size of protein or fat particles, as carbohydrates form the bulk of our food. Here, again, a starch particle is shown which can be separated into 4 segments, each representing maltose as in III. Each maltose particle is shown with 4 units as shown by its division in V. Each of these units represents a glucose particle, which is shown as 6 solid black circles representing carbon, and 6 hollow circles representing water. This illustrates the formula of glucose which is $C_6(H_2O)_6$, *i.e.*, it contains 6 molecules of carbon and 6 molecules of water.

Water, constituting 80% of our foodstuffs by weight, is represented as hollow circles scattered all over the figures, while salt is represented by the crystals shown in the figures. Vitamins are represented by a rodlike structure in the figures.

Pachaka Pitta, representing the enzymes present in the secretions of different digestive organs, is indicated by cutting instruments. Each instrument represents an enzyme. Thus:—

In II the salivary digestion shows the action of one enzyme, ptyalin, on the carbohydrates.

In III the gastric digestion showing one enzyme, pepsin, acting on the protein. The hydrochloric acid, which represents *Bhutagni*, is not shown and renin which coagulates the milk is shown at the bottom.

In IV the pancreatic digestion, three enzymes, trypsin acting on the protein, amylase acting on the carbohydrate, and liapse acting on fat are represented by three cutting instruments. Bile, which has no enzyme is shown in IV and V by the shade of background.

In V the intestinal digestion, the enzyme enterokinase acting on the protein and other enzymes splitting the carbohydrates are shown.

Pitta as mentioned before represents the glands of our body, the final outcome of which is to produce heat or energy which is necessary to maintain all functions of our body. It has been explained in the text how digestive glands in the

tion, destruction and disposal of red blood cells. The retina and lachrymal glands, the *Alochaka Pitta* regulate vision; and the glands of internal secretion, the *Sadhaka Pitta* regulate courage, fear, anger etc. (See Fig. 1A).

KAPHA

For the purpose of the study of *Kapha* the following materials are available from the *Samhitas* as indicated here by only the references. These are—

Caraka *Samhita—Sutrasthanam* — 12|12, 16; 18|40, 44; 20|18.

Susruta *Samhita—Sutrasthanam*—15|4, 15; 2|12-14.

Instead of quoting all these passages, it will suffice to mention only one—C. S. 20/18 which indicates the 20 symptoms of abnormality of *kapha* which are as follows—1. Satiety after food, 2. sleepiness, 3. increased sleep, 4. moist condition of the body, 5. feeling of heaviness of the body, 6. laziness, 7. sweet taste in the mouth, 8. increased fluid in the mouth, 9. bringing out phlegm, 10. excess of excreta, 11. lethergy, 12. feeling of pressure over the chest, 13. sticky mucus in throat, 14. fullness of vessels, 15. swelling in the front of the neck, 16. general anasarca, 17. indigestion, 18. local œdema, elephantiasis, 19. pale complexion of the whole body, 20. pale colour of the urine, fæces and eyes.

The passage of Caraka *Samhita*—C. S. 12|16 gives us a nice clue to classify all the materials refered to above. The heading of this classification is indicated thus—

1. **स्थौल्यं कार्श्यं**(general or local fullness, or œdematous condition of the body or the reverse), 2. **उत्साह, आलस्य क्लषता** and **क्लीवता, ज्ञान** and **अज्ञान, बुद्धि** and **मोह**(physical and mental fitness or the reverse), 3. **क्लेदन,** (lubricating qualities), 4. **रसम्** or **बोधम्** (qualities to ascertain the taste), 5. **दार्ढ्य** and **शैथिल्य** (uniting or disuniting the different elements of the body giving it the firmness or relaxation), and 6. **स्नेहन** or **तर्पण** (cooling qualities).

Thus the 20 symptoms enumerated above can be classified accordingly in the following way.—The symptoms numbered 4, 5, 14, 15, 16, 18, 19 and 20 may be grouped under the first heading. Symptoms numbered 2, 3, 6 and 11 under the second; numbers 1, 9, 10, 12, 13 and 17 under the third; numbers 7 and 8 under the fourth. The fifth and sixth headings are not represented here. This shows that all the manifestations of normal and abnormal *Kapha* can be very appropriately classified under the above six heads. When the first two heads are taken together it makes five groups and these exactly fit in with the description of the qualities attributed to each of the five *Kaphas, viz.* 1. *Abalambaka Kapha* (lit. which supports). This regulates general or local fullness or œdematous conditions of the body or the reverse and the physical and mental fitness or the reverse. 2. *Kledaka Kapha* (lit. that which moistens). This regulates the lubricating qualities in the body. 3. *Rasaka* or *Bodhaka Kapha* (lit. that which causes perception). This regulates the qualities to ascertain the taste, 4. *Shleshaka Kapha* (lit. that which joins). This regulates uniting or disuniting the different elements of the body giving the firmness or relaxation. 5. *Tarpaka Kapha* (lit. that which satisfies). This regulates the cooling qualities in our body.

The physiology of regulation of these functions may now be stated very briefly. 1. *Abalambaka Kapha* represents the body water of our body which pervades through every part, between and inside every cell, inside and outside every channel

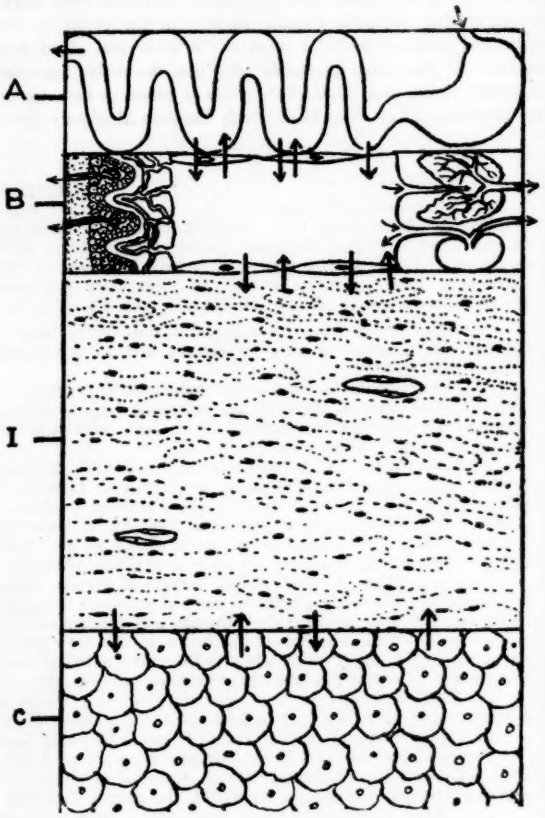

FIG. 2. SHOWING THE DISTRIBUTION OF WATER IN OUR BODY.

The figure shows four compartments marked A, B, I and C:

A—represents the alimentary canal, the source of intake of all fluids in our body constituting 70% of the total human body weight. The arrows show the entry of fluid into the stomach, partly leaving the body through the anus and the rest absorbed into the second compartment B, representing blood. In normal conditions and particularly in vomiting and diarrhœa water enters into the alimentary tract from blood.

B—representing the blood fluid comprising 5% of total body weight receives its water from the alimentary tract (A) and the interstital fluid (I) and drains away water through the skin shown to the left and through the lungs and the kidneys shown to the right of the figure. Some water is reabsorbed from the tubules back into the blood.

I—represents the interstitial fluid comprising 15% of body weight which interchanges fluid intake and output with the blood on the one hand and the intracellular fluid (C) on the other.

C—represents the intracellular fluid comprising 50% of total body weight which interchanges fluid with the interstitial fluid only.

of our body and constitutes about 70% of our body weight. This water called "Body water" exists in our body in 3 compartments. (1) Watery portion of blood (5% of body weight), (2) interstitial fluid (15% of body weight), and (3) intracellular fluid (50% of body weight). How this water interchange takes place from the alimentary tract, its chief source, to and from the 3 above named compartments, is shown in the figure 2. It is to be noted that even in extreme case of loss of fluid *e.g.* in cholera, the whole amount of blood water cannot be lost as certain amount must be there to hold the blood protein in solution. Similarly a considerable amount of intracellular fluid must remain inside the cells. This portion of water which must remain in blood and cells is known as "bound water." Constant change takes place in the interstitial fluid to keep the water balance of our body intact. An excess of this fluid constitutes œdema. The cells of our body are separate and individual units so they require some binding or supporting material to put them in groups and to have conjoint function. Further, this function can only be carried out when there is free flow of the ingrediants from within the cell outside and the reverse and that is possible when these ingrediants are in solution. The body water performs this double function by supporting the cellular elements (*abalambaka*) and helping in the functions of the cells as a vehicle in supplying the requirements and removing the efete materials. It is thus quite obvious that in abnormal conditions of this *kapha,* general and/or local fullness or œdematous condition of the body or the reverse might take place. Body fat also supports *Abalambaka Kapha* as it also suports some of our tissues but it cannot be the *abalambaka kapha* itself as it is included as *Meda Dhatu,* one of the *Dhatus* of our body. All *dhatus* support the body, as the derivative meaning of the word—*dhatu* is to support.

The other function of the *Abalambaka Kapha* is to maintain physical or mental fitness or the reverse. This may be explained thus. Body water is derived from three different sources. (*a*) from the amount of water we drink, (*b*) derived from the water contained in our foodstuffs, as water amounts to more than 80% of the total weight of the foodstuffs taken and (*c*) the water of oxidation. We are concerned with the water of oxidation here. We have already seen during the study of *Pitta* as *Agni* (fire) that the carbohydrates (glucose) during oxidation produce carbon dioxide and water as per formula $C_6H_{12}O_6+6(O_2)=6(CO_2)+6(H_2O)$ and as a result of this reaction energy or heat is produced. The water thus obtained —the water of oxidation—which is fairly considerable in amount produced as the result of oxidation of the products of digestion mainly the carbohydrates and fats is responsible for the maintenance of physical and mental fitness. When this water of oxidation produced in the tissues, accumulates locally at the site of origin, it increases the local body water as in cases of sleep during the daytime, lack of exercise, sedentary habits, partaining of too much sweets, etc. (the very causes of the production of abnormal *Kapha* according to *Ayurveda,* (S. S21/23-24). This local accumulation produces feeling of heaviness of the body, sleepiness, laziness, lethergy, increased sleep, etc.

The *Abalambaka Kapha* has still another function, *i.e.,* रोपणकृत् (S. S. 15|5) favouring the implantations of furuncles. This takes place because of the loose cellular tissue and so bacteria may have free entrance and produce the damages as the soil is ready and as no tissue response possible due to the separation of tissue spaces. The body water thus represents the *Abalambaka Kapha.*

2. The *Kledaka Kapha* represents the mucus derived from the mucous membranes of our body which moistens and lubricates—(*a*) the respiratory passages for easy respiration, (*b*) the foodstuffs to be digested, (*c*) the fæcal matter for easy expulsion. Thus we find in *Ayurveda,* abnormal *kapha* leads to vomiting of phlegm, sense of fullness of the stomach, anorexia, excess of excreta, sticky mucus in throat, feeling of pressure in the chest due to scanty or dried up mucus.

3. The *Rasaka* or *Bodhaka Kapha* has got the quality to ascertain the taste. This is the fluid which keeps the tongue moist and clear, which is the sign of health. Abnormality leads to dry tongue which is of bad prognosis. It is this moisture which is essential to the recognition of the different tastes. The actual recognition takes place by the taste-buds, the nervous structure representing *Vayu.* The fluid, moistening the tongue and helping to recognise the taste, is the *Rasaka* or *Bodhaka Kapha.*

4. The *Shleshaka Kapha* unites the different elements of the body. From the description as found in the *Samhitas* it may appear first that its function is to unite one bone with the other at the joint, thus identifying itself as the synovial fluid. On closer study, however, it reveals that the *Shleshaka Kapha* unites not only one bone to another but also muscle or tendon to bone, one band of muscle to another, the skin with the subcutaneous tissue, etc., giving the compactness of the tissue.

It appears thus, that while *Shleshaka Kapha* unites coarse with coarse structures, the *Abalambaka Kapha,* the body fluid, unites the finer and microscopical structures.

5. The *Tarpaka Kapha* cools the brain and the eye. It therefore, represents the cerebro-spinal fluid and the humours of the eyeball.

The study of Kapha may now be summarised as follows:—

Derived from श्लिष-आलिङ्गन, *Schlesma,* also called *Kapha* actually means that which joins and supports other materials. The bigger and coarse materials in our body we find in the form of bones and muscles and the union between them can be seen with naked eye as they are too coarse and big. We know that the bones, muscles and every part of our body are composed of millions of finer units called cells. Millions of bone cells form one bone as we see it, similarly millions of cells form the liver, muscles and every structure of our body. What binds them to keep them in position and thus enable them to perform a conjoint function? Each individual muscle cell contracts but the effect of contraction of only one cell will not be sufficient to be able to move a sand particle. When many millions of these cells contract simultaneously, the effect is aggregated and we find the result as detailed in Æsops Fables, in the story of the farmer and the bundle showing "unity is strength". What joins all these fine as well as the big units of our body? It is *Schleshma* or *Kapha. Abalambaka Kapha* the supporting *Kapha* as the term implies unites each individual cell of our body and acts as the vehicle for the nutrient element of our

body the *Rasa Dhatu*. *Kledaka Kapha* uniting with the hard food particles enters into their interstices thus making them soft so that *Pachaka Pitta* can act on them readily and more efficiently. *Bodhaka Kapha* uniting with the food material makes it soluble so that the teste buds of the tongue can ascertain the taste of the material thus regulating the formation of *Pachaka Pitta*. *Schleshaka Kapha* unites and lubricates the coarser elements as bone to a bone, muscle to bone, etc., *Tarpaka Kapha* sooths the brain.

THE TRIDOSA

Vayu, Pitta and *Kapha* thus functionate as the guiding principles of all the functions of our body. This can be illustrated by comparing our body with a motor car.

The driver of the motor car represents *Vayu*. He is the life of the car. Acting as *Prana Vayu* he puts the battery or magneto in action which starts the car, otherwise the car remains inactive like a dead man although provided with all parts in working order. He as *Samana Vayu* regulates the conversion by burning of petrol representing *Pitta* into energy the source of all function of the body and the car. As *Apana Vayu* he regulates the waste products and noise of combustion through the exhaust pipe and silencer. *Udan Vayu* is active through the horns and the ultimate function of the driver is to regulate the speed or motion through *Vyan Vayu*. As detailed before, the fuel, petrol represents *Pitta* which supplies heat and energy to the whole mechanism. Every part of the car requirs lubricants, water to cool the engine, bolts, nuts, etc., to keep the parts together. In the human body, these are represented by *Kapha*.

In this simile the important parts as the engine and its accessaries, the differential, wheels, chassis, etc., have no place for consideration. It may appear curious. But not actually so. All these represent the *Dhatus* or component elements of our body. The curburetter of the engine may be compared with *Rasha Dhatu*, of our body, the engine itself with *Rakta Dhatu* (blood) ; axle, differential and rear wheels which give the motion of the car as *mansha* (flesh), tyres as *Medha Dhatu* (fat), absorbing all shocks and the chassis as *Asthi* (bone). etc.

In a motor car the vital defect always depends on bad driver, bad fuel, or bad lubrication, which represent in our simile *Vayu, Pitta* and *Kapha*. As disorganisation of one or more of these three elements put the whole machinery of the car in disorder, so in the human body, disease is produced when one or more of these three elements *Vayu, Pitta* and *Kapha* are deranged. As these three are liable to be disorganised representing the human machinery they are appropriately termed *Dosas* (lit., those which are liable to be deranged). Thus *Vayu, Pitta* and *Kapha* come to be known as *Tridosa*.

This in brief is the *Tridosa* theory of Ayurveda and this aspect of the physiology has been dealt with in this monograph.

SUMMARY

The basic principle of *Ayurveda* has been expressed in a nutshell— **दोषधातुमलमूलं हि शरीरम्** *i.e.*, the essential constituents of our body consist of *Dosa, Dhatu* and *Mala*. *Dhatu* (**धारणात्** that suports) represents the combination of these units which hold or support our body. The fine and coarse substances are seven in number—*Rasa* (chyle), *Rakta* (blood), *Mansa* (flesh), *Mehda* (fat), *Asthi* (bone), *Majja* (marrow) and *Sukra* (semen). The fore-runner of these *dhatus* is the *Rasa Dhatu* from which all the other *dhatus* are derived. The *Rasa Dhatu*, again, is nothing but the assimilated part of our foodstuffs after digestion and absorption. The remaining part of the food, *i.e.*, the excreta-urine, fæces and sweat, represents the *Mala*. *Dhatus* and *malas* are thus derived from the food we take. *Dosas*, on the other hand, are the forces which regulate and control the *dhatus* and *malas*. *Vayu* (derived from **"वा"** meaning movement) is the force which gives the movement or the dynamic force to the atoms of our body. Hence movement **(सञ्चालन)** is the chief function of *Vayu* and that represents the nervous system of our body. *Pitta* is that force which as (1) *Pachaka Pitta* or *Pachaka* Fire breaks down the foreign particles, *i.e.*, our foodstuffs, into simpler body elements and causes their assimilation in the form of *Rasa Dhatu*. This is helped by the action of *Bhutagni* which leads to the physical, chemical and physico-chemical changes of our food which is composed of five *Bhuta* elements (*Panchavautic*). (2) As *Dhatwagni* breaks down the *Rasa Dhatu* inside the body still further producing heat (*Kayagni*) and energy. *Rasa Dhatu* is again broken down into the next, the *Rakta Dhatu* and so the seven *dhatus* one after the other. The *Pachaka Pitta* and the other four *pittas*, as already detailed, are the secretion of the glands of our body, of which the *Sadhaka Pitta* represents the secretion of the ductless glands. On account of these properties the chief function of *Pitta* is heat production **(स्वेदन)**.

Schleshma (lit., which joins or supports), or *Kapha* is that force which unites, supports and lubricates the finer and coarser particles of our body. Hence the chief function of *Kapha* is lubrication **(स्नेहन)**. The *Kapha* represents the body water and the watery materials of our body. The three *Dosas—Vayu, Pitta* and *Kapha* thus control and regulate all the functions of the body. These in brief, are the basic principles of the *Tridosa* theory.

JOURNAL OF THE

INDIAN MEDICAL ASSOCIATION

CALCUTTA, JANUARY, 1943

THE QUININE PROBLEM

Despite the assurances of the Government that enough quinine for two years has been stocked, every physician practising in India is finding real difficulty in getting quinine for his patients. The medical and lay papers all over India have commented on the acute shortage of this essential drug and we also have published in our November, 42 issue a thought-provoking article on the subject. The repercussion of this shortage is already in evidence in India. In our Current Comment section the interested reader will find a long correspondence on the problem with a short editorial note. All these prove one thing that there is really an acute shortage of the drug so far as the consumer is concerned whatever stock the Government may possess. The problem, therefore, resolves into an investigation of the causes leading to this state of affairs and finding ways and means to solve those difficulties.

Whenever we think of quinine problem we are to face the fact that only about one third amount of actual consumption (200,000 lbs.: 70,000 lbs.) were being produced in India, though we could produce ten times the amount than is required by us.[1] We had the occasion in the past to point out how the India Government joined hands with the Anglo-Dutch concern the Kina Bureau and the English firm Howards'. Dr. K. S. Ray, in the Presidential address (1941) quoted Krishnan[2], who had asked why could not we be self-sufficient when so much opportunities were present. The answer was of course the quinine monopoly. The short-sighted policy of the Government or rather the selfish motive of the foreign capitalists have thrown the country into such a chaotic and tragic condition as we find to-day. This is the first and the main cause of quinine shortage in India. But this is not all. Strange things have been happening behind the screen. The tragedy of the thing lies in the fact that when we were making our voice hoarse crying for more quinine, the acreage under cinchona had been steadily declining with persistent export of cinchona bark. The following tables are significant:

DECLINE IN CINCHONA ACREAGE IN INDIA
PROVINCE BY PROVINCE

Year	Bengal	Burma	Madras	Total Br. Provinces
1932-33 ..	2,600	1,150	2,081	5,831
1933-34 ..	3,100	465	1,624	5,192
1934-35 ..	2,500	588	1,630	4,718
1935-36 ..	2,600	142	1,618	4,360
1936-37 ..	2,700	?	1,614	4,314

EXPORT OF CINCHONA BARK FROM INDIA
(ALMOST WHOLLY FROM MADRAS)

	1934-35	1935-36	1936-37	1937-38	1938-39
Quantity in lbs.	1,41,798	24,118	52,231	28,222	32,452
Value in Rs.	42,371	6,302	16,026	9,820	11,117

This steady decline in cinchona acreage must be checked and further, a saner export policy must be formulated for the future.

The reason is not far to seek. In the opinion of Chopra this was due to the Kina policy. He observes[3]: "The Kina Bureau has tried and has been successful in effecting regulated and gradual reduction of the cinchona areas to proportions fitted to what the world can afford to buy and not what it really needs."

Furthermore, while by scientific methods, suitable weeding and selection of ground, Java bark was kept at a high-yield standard, the quinine in the Indian bark was allowed to deteriorate and no appreciable effort was made to raise its standard by scientific methods. We have remarked in the past that owing to a guarantee given to the Kina Bureau and Howards', India was forced to buy quite a large amount of quinine from outside, with consequent accumulation of stock of quinine in the Central Government Stores. The question which naturally arises is where is the stock? Why more of it is not released for the civilian population? Why the profession and the public should not get their proper quota of quinine?

Over and above what the Government used to buy foreign quinine as a minimum guarantee, private individuals—drug houses, businessmen, dispensers used to import quite a lot more of quinine from Roche, Howard, Merck and others. These were mainly sold to ordinary

1. *J. I. M. A.*—Editorial, 11, 280, 1942.
2. Krishnan quoted by Ray—*J. I. M. A.*, 10, 194, 1941.
3. Current Science—Editorial, 11, 349, 1942.

dispensaries for preparing antimalarial patent medicines and contraceptives. It is quite possible that a large store of the drug is still in the stock of the profiteers. Anybody who keeps his eyes open will be astounded to find that even people in streets of big cities like Calcutta are either selling or buying quinine from black market at very high rates. We wonder why the Government do not freeze this stock, regulate the price and release it for civilian consumption.

The quinine position in the country being what it is now, what is the solution? The Government should take the public and the profession into their confidence and explain the real situation and how they wish to meet the problem. Advice should be sought from the public and a permanent ad hoc committee should be formed to advise the Government on the different aspects of the question. The situation has already become serious and it is time that the Government woke up from their usual sloth and slumber and take a long view of the question and has to find permanent ways and means for the betterment of the health of the public. We must put our heads together and find out ways and means to get out of this impasse. Naturally we are very much interested in Ghosh and Roy's article on the subject[4]. We again draw the attention of our readers to the "Current Comment" published this month, where we have given the whole correspondence on the subject published in a lay paper together with our comments and League of Nations recommendations. Dr. E. G. H. Koenigsfeld has written an interesting letter on the subject for which we would refer the reader to this months' correspondence column. All these demand careful consideration by both medical men and the Government.

Today when we are facing the impossible and unequal fight with a deadly and devastating disease, the first thing that comes to our mind is whether we can prevent the disease in an effective way without quinine. We agree with Grant that unless the economic condition of the country becomes considerably improved, we can never hope to prevent malaria with cash money. Hence all *immediate* preventive schemes for malaria must pay special heed to the alternative methods of prevention. We think that with considerable impenetration of the soldiers in remotest villages of India, the military authorities should necessarily be interested in the public health and antimalarial problem of the country. If the soldiers are entrusted with the public health ideas by their doctors and they lead in taking a hand, which they should, we are sure that the civilian population will come forward with their mite. In the meantime the Public Health Departments of the Government, Municipalities, District Boards and the Departments of Irrigation and Engineering should be aroused from their century-old slumber and asked to work day and night in full co-operation of the population. The present War is sure to last longer than what some of the optimists think before final victory is won. When the fullest understanding between the civil, military and public health authorities is reached, much improvement can be made in the highly endemic areas in 2 or 3 years. Our country can ill afford the colossal economic loss due to incapacity of the malarial patient to work, cost of treatment, indirect effects like easy vulnerability to tuberculosis, leprosy, etc., especially during the War. Incidentally, it may be mentioned that the probable incidence of this preventable disease in the province of Bengal is between 30 to 40 million cases per year out of a total population of 51 millions. A staggering figure indeed! The face of the Indian villages need be changed. Tube wells should be sunk. Swamps, pools and puddles filled up and better irrigation system introduced associated with other intensive antimalarial measures. Like China, the people of India can march out for conquering malaria.

Prevention of malaria is the best War effort that we can think our Government can aim at at present. To protect our country we require healthy, hardy people of the soil. A rustic civilian of to-day may be the soldier of to-morrow fighting for freedom and defence of his own country.

When we come to therapeusis, our course seems to be quite plain. First and foremost, we must avoid wastage in prescription, contraceptives, restriction of other prophylactic use and so on. But we think further saving can be made if quinine mixtures are stopped in well equipped hospitals of district towns and subdivisions and intramuscular injections are resorted to. The amount of quinine required by injection is surely much less than is taken by mouth. A considerable amount of saving can be done in this way. Unfortunately, this cannot be advocated for ordinary dispensary practice, where the patient shall have to go away after taking the injection. Rest is essential, which

4. *J. I. M. A.*—12, 46, 1942.

we can hardly have there. Any way, these procedures will surely save much quinine for our use. The second method of solving this problem will be using the quinine substitutes and other antimalarial drugs, indigenous or otherwise.

We are happy to find that mepacrine hydrochloride is being prepared in this country. Government can do much to help the preparation of the drug in the country by subsidising companies, encouraging research for other substitutes, releasing drugs and machineries required for its preparation and looking after standardisation of the quality and toxicity of the drug. This will certainly lessen the quota of quinine required by us. Cinchona febrifuge, that neglected but old drug should be pushed more. It has been found that the other alkaloids of the cinchona bark have got also certain antimalarial properties. As Chopra[5] points out "it is very far from certain that quinine is the best alkaloid of cinchona bark to use. Both quinidine and cinchonidine are more efficacious with regard to their antimalarial power."

Koenigsfeld has suggested chestnut extract as a substitute for quinine. Lastly, indigenous antimalarials should be scientifically analysed, tested and tried both *in vivo* and *in vitro* to see whether they can conveniently replace quinine. Capt. G. Srinivasa Murthy[6] has revealed that during the days of the East India Company, a number of indigenous antimalarials were "authorised as official substitutes for cinchona and its alkaloids. They were also included in the pharmacopœia of India which was then in use as supplement to the British Pharmacopœia."

In conclusion, we would point out again that the present perilous situation is the outcome of the short-sighted policy of the Government, who could have ten times our requirement if they would not have been a party to the exploitation of world market by the Kina Bureau and it is up to the Government to remedy this by immediately working up machineries to make the health of the people as secure as possible.

5. Current Science—Editorial, 11, 350, 1942.
6. Current Science—Editorial, 11, 349, 1942.

SECOND BIHAR PROVINCIAL MEDICAL CONFERENCE

The Second Bihar Provincial Medical Conference will be held at Chapra (Saran) on the 24th of January, 1943.

There will be a scientific section also in connection with the Conference. Abstracts of papers along with the original whenever possible, should reach Captain M. Hussain, M.B., F.R.C.S., D.L.O., D.O.M.S., Professor of Ophthalmology, P. W. Medical College, Patna, Chairman of the Scientific Section by the 15th of January, 1943.

PROGRAMME

24th January, 1943.

9-00 A.M.—Annual General Meeting of the Bihar Provincial Branch.
10-30 A.M.—First Meeting of the Bihar Provincial Council and of the Subjects Committee.
2-00 P.M.—Bihar Provincial Medical Conference (Open Session).
5-30 P.M.—Group Photograph followed by Tea by Messrs Oriental Chemical Works, Calcutta.
7-30 P.M.—Scientific Section.

CURRENT COMMENT

QUININE PROBLEM

Many of our readers may have read with interest in the columns of the *Statesman* on "Quinine Shortage" by Lt. Col. A. C. MacGilchrist, a retired member of the Indian Medical Service and a former professor of Physiology at the Calcutta Medical College, and rejoinders to Col. MacGilchrist's letter by Dr. B. Mukerji, Officiating Director, Biochemical Standardization Laboratory, Government of India, Dr. C. Strickland, Malariologist, Army in Burma and late Officiating Director, School of Tropical Medicine, Dr. P. L. C. Carrier, Chief of Medical Officer, Burma Railways, etc. Col. MacGilchrist made a plea that 5 grs. quinine per day (?) was sufficient to treat malaria and strongly criticized the method of quinine administration by the intramuscular route, characterising it as wastage of quinine'. This argument is so frankly anomalous and unsupported by modern malariologists that comment is unnecessary. The same applies to statements by Dr. C. Strickland quoted below, "Let the patient run his fever for 3 or 4 days without quinine; (2) then giving him say 5 to 10 grs. in all, and (3) if he is not then well, treat the soil and not the seed, the patient and not the infection—*i.e.,* give him tonics, and *if necessary send him to the Hills or Home.*"

Dr. Strickland was apparently day-dreaming and inspite of many years of service in India and Burma, he appears to be unware of the fact that there are four hundred million inhabitants of India who cannot be treated by sending them to the 'Hills' and they cannot be sent 'Home' as their home is India and not England.

We are giving below the correspondence referred to above. These are illuminating and self-explanatory. The letter of Dr. Carrier sums up the position admirably.

In consideration of the importance of the subject to our readers who may not have ready access to the authoritative publications by the League of Nations on the subject, an abstract from the Malaria Commission's conclusions is annexed. This will help them to form a clear picture of the present position on 'Treatment and Prophylaxis of Malaria'.

We would also refer our readers to the Editorial Comments on the subject this month.—EDITOR.

SHORTAGE OF QUININE

Letter dated Calcutta the 16th August, 1942, from Lt.-Col. A. C. MacGilchrist to the Editor, The Statesman.

Sir, In connexion with the new order on the above subject, dated London, August 12, referred to by you lately, it is doubtful if there would be any shortage if doctors (1) did not prescribe unnecessary large doses (2) did not waste quinine by administering intramuscular injections.

For 30 years I have never prescribed a larger dose of quinine than five grains for an adult; and I have never been disappointed with the therapeutic results. Doses larger than five grains are not only unnecessary but *may* injure the patient. I have met with quinine deafness among doctors themselves.

Intramuscular injections of quinine are not only useless waste but cause serious physical injury by destroying all muscular tissue which comes in contact with the quinine solution. The quinine thus injected is chemically altered and of no therapeutic value.—Yours etc.

* * * * *

Letter dated Calcutta the 28th August, 1942, from Dr. B. Mukerji, Biochemical Standardization Laboratory, to the Editor, The Statesman.

Sir, I have read the letter by Lt.-Col. A. C. MacGilchrist. As some of the statements therein are not borne out by authoritative literature, it seems desirable to add a rejoinder.

Col. MacGilchrist has drawn inspiration from his *personal* experience. While he can speak with a certain authority on quinine, having carried out significant researches in this field, he is either unware of or has overlooked later developments by such workers as Rogers, Megaw, Acton, etc., in India, Manson-Bahr in London and by the Malaria Commission of the League of Nations. It is now generally recognized that *at least* 15 grs. of quinine by the oral route are needed for 5-7 days to counteract an attack of malaria. Col. MacGilchrist is not clear as to the total amount of quinine he would recommend per day in adults; 5 grs. once a day is certainly not going to do the trick.

Intramuscular injection, while, not a method of choice in malaria therapy, and while frequently abused, has a place in particular cases. Any medical practitioner in the rural areas would testify to this.

There is authoritative evidence that quinine is absorbed from the site of injection (intramuscular) and exercises a well-marked therapeutic effect. Quinine, through whatever route administered, is partially destroyed in the liver but about half can be recovered unchanged in the urine. Wastage must be prevented, as the quinine shortage is bound to be more keenly felt, but decrying intramuscular injection of quinine as "wastage" and recommending its administration only in 5 grain doses per day (?) is certainly not the royal road for achieving the desired goal.—Yours etc.

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Letter dated Calcutta the 1st September, 1942, from Dr. C. Strickland, Malariologist, Army in Burma, Late Professor and Officiating Director, School of Tropical Medicine, Calcutta, to the Editor, The Statesman.

Sir, With reference to the letters from Lt.-Col. MacGilchrist and Dr. B. Mukerji, may I add the following observations? When the Dutch physicians, observing the effect of quinine on malaria-injection induced for the treatment of certain venereal conditions, state (1) that after the initial reaction of the patient to the fever (say 3 or 4 days) a single dose of quinine (if I remember rightly, they say 5 grains) suffices to stop the fever; and (2) if this does not suffice no amount will do any better, then I think their theory should be carefully examined.

It explains much, for instance (1) the number of opinions among practising doctors as to the correct procedure of giving quinine—their methods vary so much because they are all wrong; (2) the number of malaria cases on whom quinine has no effect; (3) the fact that in West Africa in the early days it was the practice to let the patient run his fever for few days before giving him quinine, a practice which the League of Nations Health Commission has tentatively endorsed; (4) that during the last War, a country doctor at Home reported having seen his first case of malaria, one Home from the Front, and that having cured it with "heroic doses of quinine, one grain three times a day", he was laughed at, but he was near the

truth; (5) that whereas sometimes quinine does not cure a patient, a visit Home or to the Hills has the desired effect.

Anyway, clinical men are tending to Col. MacGilchrist's contention. We have left behind the days when an eminent physician, on rational grounds, gave 80 grains a day.

The same rationalist also observing that sweating was beneficial in malaria had an oven constructed in his back-garden and roasted his patients.

And in spite of my friend Dr. Mukerji, a meeting of nearly all of the leading "tropical specialists" at Home in the rooms of the Royal Society of Tropical Medicine on June 18, 1925, endorsed the opinion, without a dissentient, that 3 day courses of "moderate dosage" sufficed to cure, and no greater dosage or other variation in the procedure made any difference.

It would, therefore, seem worth while to follow the teachings of those eminent Dutch physicians, and adopt the practice of: (1) letting the patient run his fever for 3 or 4 days without quinine; (2) then giving him say 5 to 10 grains in all, and (3) if he is not then well, treat the soil and not the seed, the patient and not the infection, *i.e.*, give him tonics, and if necessary send him to the Hills or Home.

This procedure would save a lot of quinine, and we would hear less of the danger of shortage during the tremendous epidemics that we are likely to have in the not distant future. Yours etc.

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Letter dated Calcutta the 2nd September, 1942, from Lt.-Col. A. C. MacGilchrist, I.M.S.(R.), in reply to Dr. Mukerji's rejoinder (vide supra), to the Editor, The Statesman.

Sir, A reply to Dr. Mukerji's rejoinder to my letter is called for.

No statement was made in my letter which is not supported by authoritative literature. His mentioning the name of Rogers, Megaw, Acton and Manson-Bahr is useless. Not one of these gentlemen ever had any practical training in pharmacology and can be taken as an authority on a pharmacological subject. To quote Dr. Mukerji—"it is now generally recognized that *at least* 15 grains of quinine by the oral route are needed for 5-7 days to counteract an attack of malaria." As this differs from my statement only in his *at least* to my *at most*, Dr. Mukerji's rejoinder is merely a task of supererogation. My point is that a maximum dose of 5 grs. thrice daily is the oral treatment for malaria and, I may now add, that this treatment will break the fever in four days, if the fever is malarial. To prevent any further misunderstanding I do not say that 4 days' treatment completes the cure.

When I first arrived in India (1900), I found that the dose of quinine universally employed in India was 10-20 grains, although the British Pharmacopœial dose was 3-5 grains. Big doses are not yet extinct; hence my letter.

As regards intramuscular quinine injections, there is ample authoritative evidence of grave damage to muscle tissue. Dr. Mukerji wisely avoids contradicting this statement. A good doctor sees to it that his treatment, if it does not help the patient, will at least not injure him. Some doctors will continue to employ brutal disabling and therapeutically useless intramuscular quinine injections until one of his fraternity is hauled for damages in a court of law.—Yours etc.

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Letter dated Dehra Dun the 4th September, 1942, from Major J. R. Foy, I.M.D., to the Editor, The Statesman.

Sir, Whatever may be the pros and cons regarding intramuscular injections of quinine for the cure of malaria, there is no gainsaying that it is not only economic and rapid in effect but also prevents the loss of many working hours to the employer and the employee.

A favourable side of the controversy regarding the "dangers" of intramuscular injection seems due after the courtesy you extended to Lt. Col. MacGilchrist's adverse opinion.

In the *British Medical Journal* of the 8th March, 1941, Dr. R. H. Hall of Kano in Nigeria states that he has been giving three daily intramuscular injections of quinine as a routine measure in his 300 bed hospital at Kano for years; without any ill effects whatever in either males or females.

I have given a few thousand intramuscular injections of quinine during the past 35 years, and have never had an abscess, or in females an abortion or miscarriage.

In 1904-5 a severe epidemic of malaria broke out among a unit at Rawalpindi. Quinine by mouth failed to control it; relapses were frequent; a very large proportion of the regiment was unfit for duty.

Each man of the regiment was given six ten-grain intramuscular injections of quinine, the first three on consecutive days and the next three at intervals of three days.

I never had any abscess, and the injected men were back to duty within a fortnight.—Yours etc.

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Editorial Comment, Amrita Bazar Patrika, dated the 8th September, 1942.

Two doctors, one an Indian and the other a Britisher, have entered upon a controversy over the manner in which quinine should be prescribed in malarial cases, particularly in view of the shortage of this drug. This might interest the members of the medical profession but not laymen. What the latter want is that quinine should be made available at a cheap price. We were assured by the Government of India that India still possessed two and a half year's stocks of quinine. Well, that is poor consolation to the suffering public. Their grievance is, and that is perhaps also the grievance of physicians and druggists, that quinine is not easily obtainable and that its price has gone up very high. It is time the grievances were redressed.

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Letter dated Simla the 12th September, 1942, from Dr. P. L. C. Carrier, Chief Medical Officer, Burma Railways, to the Editor, The Statesman.

Sir, The views of your correspondents seem to need clarification and criticism.

Col. MacGilchrist considers there would be no shortage if doctors (*i*) did not prescribe unnecessarily large doses, and (*ii*) while intramuscular injections are rightly deprecated, he would be on safer ground if his only reason were the undesirable but purely local effects at site of injection rather than the alleged inefficacy of absorption, for the excretion rate at least is equal whether the drug is given by mouth, vein or muscle.

Dr. Mukerji doubts the therapeutic value of a daily dosage of five grains, and so do I, but it seems to me the real point has

been missed, for dosage must depend on very varying conditions and Col. MacGilchrist is advocating standardizing dosage without considering the variable factors.

We do not know how quinine acts on the malarial parasite though from early days we were taught that quinine was a general protoplasmic poison and lethal to Protozoa (but not all). The generally accepted teaching is that it does not act on the parasite itself while *in* the blood corpuscle, but only when free among the corpuscles. There is, however, no convincing evidence and it is doubtful whether quinine has any *direct* action on the parasite. It appears to have an *indirect* action in some way (probably with the aid of the body's defensive tissue mechanism) that "inhibits" the parasite, or as Dr. Mukerji puts it, "does the trick". Even with the usual maximal doses (thirty grains daily, the concentration of quinine in the blood does not exceed one in a hundred thousand and Kirschbaum has shown that parasites incubated in blood containing as much as one in ten thousand part of quinine will still produce malaria if injected into man. So in the test tube at any rate the usual concentration does not kill malaria parasites.

Major Strickland refers to the Dutch School and experiments with "induced" malaria showing that a single dose of 5 grains of quinine "terminated the fever and that if it did not do so, no amount would do any better." This is difficult to credit.

Anyhow these experiments were with infections induced for purposes of treatment of certain types of nervous diseases and the point not mentioned is that malaria thus induced is not quite the same thing at any rate in response to treatment, as the ordinary type in patients suffering from malaria *ab initio*. Yorke, in his classical experiments found that administration of 15 grains of quinine for several days in artificially induced cases promptly checked the fever and only 2 per cent relapsed subsequently, but the same treatment of malaria patients who furnished the parasites for the experiments gave strikingly less favourable results with relapse rate of sixty per cent. This might be because in artificially induced cases the infection is *recent* and much more amenable to quinine than in *well established* or old cases. Hence, dosage based on experiments with this variety of "induced" malaria is no guide for the average malaria case in endemic areas.

Ross (1919) in two thousand four hundred cases showed that of those untreated, seventy-five per cent relapsed within a month, but seven grains of quinine daily to others reduced the rate to ten per cent a month, Administration of three grains daily was less effective than seven grains, and fifteen grains daily were apparently no more effective than seven, though more noxious to the patient. Also a group receiving sixty grains daily for two-three weeks was much less successful than those receiving smaller doses and a relapse rate of thirty-five per cent occurred in this group.

These investigations not only lead one to the conclusion that if a standard dose is adopted for all cases, it should be between the two extremes, but raises the question what constitutes a "cure". To some this means cessation of fever, to others clearance of the parasite from the blood in addition; but both these events usually occur, yet the patient subsequently has a relapse, and this is of course the real criterion.

The efficiency of approximate dosage must be assessed, therefore, according to what one's standards of "cure" are; whether the infection was "recent" or long standing, what was the particular variety or form of parasite, since some varieties of parasite or forms of the same parasite are more quinineresistant, or less vulnerable from differing causes, than others.

To conserve supplies of quinine at the expense of sufferers or at the price of extending the infection by increasing reservoirs of malaria carriers by incompletely treated cases, is neither ethical nor economical, and dosage, method of administration, as well as the choice of other antimalarial preparations should be varied to meet "all comers."

I find it difficult to comment upon Major Strickland's suggestion in his penultimate paragraph, namely to follow the Dutch School in (a) "Letting the patient run his fever for three-four days without quinine," and (b) "Giving him five to ten grains of quinine in all."

This conception is attractive, the idea being attempted establishment of immunity by the body tissues on the principle of "letting the dog see the rabbit." If it worked well in malaria the possibilities would be astounding and might in time alter the history of the world, but at best it could only work in what has been described above as very recent cases and like so many of its brilliant predecessors, the idea conceived in the laboratory proves disappointing in the real test, and that particular hare just "won't run"—at any rate in the direction that those of us who have tried it would wish.

If tentatively endorsed by the League of Nations Health Commission, it must have been with reservations expressed or implied. That august body certainly did not subscribe to(b) on the contrary, if my memory serves me aright, it recommended that if standard treatment was adopted it should be a short course of fifteen grains of the drug daily in five divided doses for seven days, this to aply also to relapses; Meantime the suggestions of the Dutch School referred to above should be disregarded for practical purposes, for in that direction, amongst other dangers, lies Cerebral Malaria; and that one is a KILLER.

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Therapeutics and Prophylaxis of Malaria. Conclusions of Malaria Commission of the League of Nations.

In 1935 the Malaria Commission of the League of Nations arranged for experiments to be made on the treatment and prophylaxis of malaria with synthetic drugs and with quinine so as to compare their efficacy. These experiments were conducted according to a plan which had been prepared beforehand. They covered 12,288 subjects and were carried out in Algeria, Italy, the Federated Malay States, Rumania and the Union of Socialist Soviet Republics under the direction of Prof. Edmond Sergent, Professor Bastianelli, Dr. Neave Kingsbury, Professor Ciuca and Professor Serguieff, respectively.

The commission has drawn up the text of its fourth general report on the basis of lessons to be learned from these experiments. It represents the unanimous views of the commission and has since been incorporated in the Bulletin of the Health Organization.

The following are the practical suggestions and conclusions of the report regarding the treatment and prophylaxis of malaria:—

Without presuming to lay down hard and fast rules, the commission believes that it is in a position to give certain indications. Account should, however, first be taken of the following

points, (on which reservations have been made), with respect not only to the individual treatment of patients but generally also to the application of any therapeutic procedure: (1) the varying reactions of the different parasite strains of the same species and of patients to the drugs; (2) the special indications applicable to the parenteral administration of drugs; (3) the drawbacks of the synthetic products (yellow) coloration of the skin by atabrine, toxicity of plasmochin).

Individual Treatment of Patients: It is always desirable that the doctor should be in a position to diagnose malaria and to determine the species of parasite concerned, by a microscopic examination of the blood.

In ordinary cases of *P. vivax* (benign tertian) infections, it is almost immaterial whether quinine or atabrine is employed for treatment of the attack. Plasmochin, associated with quinine or atabrine or administered after these drugs, has no appreciably useful effect on the attaks but seems to reduce the frequency of subsequent relapses.

The association of plasmochin with quinine, or its administration after atabrine, is useful in *P. falciparum* infections, on account of its action on gametocytes and relapses.

Treatment in the Field: Atabrine, when used for collective treatment in daily doses of 0·3 gm. (for adults) for from five to seven days, acts in the same *way as quinine in daily doses of from 1 to 1·3 gm for from five to seven days or more.* There is no reason save financial considerations why either quinine or atabrine should be preferred. The manner in which collective treatment is conducted will depend on the intensity of the endemicity, which is itself the resultant of a series of factors; the incidence of malignant tertian infection (*P. falciparum*), the virulence of the strains, the sensitiveness of the strains to the various drugs and the susceptibility of various population groups.

Collective treatment with quinine or atabrine may usefully be accompanied or followed by plasmochin treatment, to diminish the number of gametocytes and the risk of relapses.

The choice of basic drug for collective treatment should be left to the public administrations or malariologic organizations that undertake or control such treatment and will be guided by local and economic considerations as well as by the preferences of the medical profession and of the population. It should be remembered that the choice of drug, as well as dosage and duration of therapeutic administration, should, so far as possible, be directed toward the achievement of the *real aim of mass treatment. This aim is to secure the largest number of complete cures in case of malaria and to reduce to a minimum the risk of anopheline infection either by direct action on the gametocytes or by indirect action on the parasites generally,* thereby effecting an eventual reduction in the number of carriers of sexual forms (gametocyte therapy). It would therefore be wise not to rely on plasmochin alone for this second purpose. These considerations also apply to the choice of the basic drug and the manner of its administration to the community either directly associated with or followed by plasmochin.

There are, however, large malarial areas, especially in the tropics where such treatment is impossible of practical application for various reasons, often financial. Under such conditions it is desirable to provide adequate and easily available treatment for the clinical manifestations of the disease, so that the morbidity and the physical incapacitation of the afflicted persons are diminished. Such treatment has very often to be placed in the hands of laymen for distribution and little or no direct medical supervision is possible. In these circumstances, the cinchona alkaloids appear to be the more suitable drugs.

Mass Drug Prophylaxis: This has a twofold purpose: (1) to protect the population undergoing prophylactic treatment from the clinical manifestations of endemic malaria, in order that its working capacity and comparative standard of health may be safeguarded without injury or its preimmunization, even in areas in which it is exposed to repeated reinfection; (2) to reduce in due course, the sources from which the local mosquitoes may be infected.

No prophylactic method, unless applied to disciplined communities under stringent supervision, is capable as yet of attaining these two objects. Special stress should be laid on the desirability and the difficulty of promoting the immunization process in the population, which depends on the degree of tolerance to infection and on repeated reinfection. At the same time one wishes to avoid the risks attendant on the presence and persistence of such latent infections in the community.

Experience has shown, at all events that useful results can be obtained with daily doses of quinine (0·4 gm.) administered during the whole of the malaria transmission season and even for a few weeks longer. This is also true of bi-weekly doses of atabrine (from 0·2 to 0·4 gm. a week) administered in certain conditions. With the latter method (biweekly atabrine), which has given encouraging results, further trials would be desirable. The daily dose of 0·05 gm. of atabrine recommended for prophylactic purposes has proved inadequate. The harmlessness of quinine makes it a suitable drug for administration by subordinate personnel without constant medical supervision, whereas such supervision is essential in the case of atabrine.

Plasmochin should not be distributed for prophylactic treatment otherwise than under direct medical control. Its use in mass prophylaxis would be justifiable only if administered to a disciplined population, which should be kept under constant medical care. It is useful more especially for the purpose of ultimately reducing the number of gametocyte carriers and arresting the transmission of infection to the anopheles. Plasmochin is certainly the gametocidal agent par excellence especially where *P. falciparum* is concerned. But both quinine and atabrine also exercise in the latter case a gametocidal action by destroying the sexual forms in process of development.

The real efficiency of such methods in the field is, moreover largely dependent on a highly important epidemiologic factor; namely, the children. The proportion of gametocyte carriers is much higher among children than among adults, given the same environmental conditions (village, house, family) in respect of endemicity and anophelism. Children, on the other hand, are more difficult to subject to regular treatment, and finally the doses and form of administration of atabrine and plasmochin to children cannot be regarded as finally settled.

Drug Eradication: Experience has so far shown that the eradication of malaria from a locality by the curative and prophylactic treatment with the drugs at present available is practically impossible. It is impossible to reach, in sufficient time, all the inhabitants of an area or even of small village. Moreover, while curative and prophylactic treatment may greatly diminish the morbidity, it cannot suppress the parasites in all the carriers.

CURRENT MEDICAL LITERATURE

MEDICINE

Roentgenoscopy as a Diagnostic Aid in Coronary Occlusion

Master (*Am. J. Rœntgenol.*, 1941:45:350) gives in the following lines the summary of his rœntgenoscopic observations of ventricular contraction in 300 private patients more than half of which had coronary occlusion and the remainder included other types of heart disease and normal subjects: The technique employed is described in detail. Rœntgenoscopy is shown to be a simple and inexpensive method of diagnosing coronary occlusion with myocardiac infarction. Abnormalities in pulsation were present in 75 per cent of the cases of coronary occlusion. Systolic expansion (reversal of pulsation) of the left ventricle observed in 50 per cent of these cases, is characteristic, if not, pathognomic of myocardial infarction. Lag and doubling of pulsation are incomplete forms of systolic expansion. Systolic expansion is seen in practically every case of large heart with ventricular aneurism. Absence and diminution of pulsation were present in 25 per cent of cases with coronary occlusion but also occurred in other types of heart disease. The abnormal pulsations observed were located in the apical and supraapical portions of the left ventricle in almost 85 per cent of the cases studied. The incidence of abnormal pulsation in coronary occlusion was found to be greater when the heart was enlarged. When the area of abnormal pulsation was large the prognosis was poor.

Systolic expansion may appear directly after the coronary occlusion and persist for many years. Its disappearance or a change to absence or diminution of pulsation is of favourable significance.

In normal hearts the pulsations are always normal. Hypertension alone does not produce changes. Disease of the coronary artery, without occlusion, did not exhibit systolic expansion or absence of pulsation. When these conditions are present coronary occlusion should be suspected. Systolic expansion is seen occasionally in cases of aortic insufficiency with cardiac enlargement.

Rœntgenoscopy should form part of the routine examination of every cardiac suspect. It may be positive when the physical examination and electrocardiogram are negative.

Effect of Pentobarbital on the Cardiac Conduction System*

van Dellen and others (*Am. Heart J.*, 1942, June) report on the study of the effect of pentobarbital and digitalis on the cardiac conduction system of dogs by means of the electrocardiogram. Pentobarbital was found to alter the function of the conduction system in the digitalized dog. Changes produced by digitalis, such as bradycardia, partial or complete auriculoventricular heart block, and ectopic impulses of ventricular origin, were abolished in five dogs by pentobarbital. In five other dogs pentobarbital produced ectopic tachycardia of ventricular origin. When the ectopic tachycardia of ventricular origin was produced initially by digitalis, pentobarbital had no effect except to increase the auricular rate. When digitalis is given to the dog which is anesthetized with pentobarbital, the usual digitalis response may not be elicited. The action of pentobarbital on the conduction system is probably the result of its depressing effect on the vagus.

The Determination of Local Anaesthetic Potency by Observation of Nerve Action Potentials*

Bennet and others (*J. Pharmacol. & Exper. Therap.*, 1942, June) report on the observations of the effects of local anæsthetics upon isolated frog sciatic nerves as indicated by changes in the alpha wave of the action potential. They developed an apparatus for this purpose and an empirical equation from which the relative potency of local anæsthetics may be rapidly obtained under controlled conditions. The block-time for a given nerve has been shown within limits to be directly related to the square of the diameter of that nerve. They have demonstrated that for all local anæsthetics investigated the block-time for nerves varies inversely with the logarithm of the molar concentration.

Effect of Alkalosis and Acidosis on Cortical Electrical Activity and Blood Flow*

Lubin and Price (*J. Neurophysiol.*, 1942, June) report on the study on cats, involving observations on the electrical activity of the cortex following acidosis or alkalosis. They found that the minimal amount of acid (HCl) or alkali (Na_2CO_3) injected intravenously necessary to produce changes in the respiratory rate of the anæsthetized cat does not disrupt the cortical potentials. Changes in the electrical activity of the cortex following acidosis or alkalosis occur only if near-fatal amounts are injected or, in the case of alkalosis, if an abnormal pattern has already been established as in insulin hypoglycæmia. While intravenous injection of acid causes dilatation of the pial arteries in most cases, and intravenous injection of alkali causes constriction in most cases, these changes are not obviously related to the alterations of cortical potentials.

Hypoglycaemia

Laviates and Peters (*International Clinics*, Ref. *Med. World*, 1942:57:36) write that nocturnal manifestation of hypoglycæmia which is most frequently encountered is a headache or a sensation of pressure over the top of the head; in other cases restlessness and perspiration during the night may be the first indication of the onset of hypoglycæmia. There may be in other patients tremulousness, confusion and weakness complained of before breakfast. .In fact any new symptom appearing between bedtime and breakfast should be regarded as an indication for the reduction of the dose of insulin. The common early symptoms met with during the day time are hunger, nervousness, blurring of vision, want of concentration power, confusion and sweating. Less commonly one meets with vomiting, double vision, numbness of the finger tips and numbness and thickness of the lips and tongue. The authors conclude with the statement that the clinical importance of deglycogenation produced by over-insulinisation is not sufficiently appreciated.

Dysentery Carriers Treated with Sulphaguanidine

Rantz and Kirby (*J.A.M.A.*, 1942:118:1268) write that the drug has been experimentally found effective in inhibiting the growth of various dysentery bacilli and is clinically successful in the treatment of acute dysentery. This has made the authors

try the drug in 10 members of the staff of a hospital who harboured desentery bacilli. They were all ambulant and were regarded as healthy carriers but a few had had rather non-specific gastrointestinal symptoms, such as abdominal cramps, and diarrhœa. Each of them were given 12 gms. of sulphaguanidine daily for 2 to 6 days, the total amount varying from 16 to 84 gms. Sulphaguanidine was still demonstrable in the stools for several days after its administration ceased. After this treatment dysentery bacilli disappeared from the stools in 9 out of 11 cases; in the remaining 2 cases repeated and larger doses of the drug failed to eradicate the infection; the organisms disappeared for a few days but returned soon after. Apart from slight headache the drug caused no toxic disturbances and all the patients remained at work throughout the course.

Intravenous Therapy with Invert Sugar

Keer and Panly (*Surg., Gynec. & Obstet.*, 1942:74:925) write that during the last war owing to the difficulty of obtaining glucose invert sugar was used for intravenous therapy with success. They have again found it necessary to use it. The solution is prepared from ordinary cane sugar by rapidly hydrolysing with hydrochloric acid, then neutralising with sodium hydroxide. The pH is adjusted and after filtration the solution is made upto 5 per cent. Full details of the method of preparation are given. Invert sugar contains both glucose and levulose and there is ample evidence that the body can properly convert the levulose into glucose and glycogen. The small amount of unhydrolysed sucrose which may remain in the invert sugar can be rapidly excreted in the urine. The results are highly satisfactory. Only 3 reactions occurred amongst the first 6,180 cases treated with invert sugar and none in the later series of 2,349 cases. The authors conclude that when glucose is not available invert sugar is a satisfactory substitute for use in the intravenous therapy.

Bacteriostatic Effect of Sulphathiazole in Ointment Bases*

Strakosch and Olsen (*Arch. Dermat. & Syph.*, 1942, July) write that the management of streptococcic and staphylococcic infections of the skin presents an important problem. They reported on the studies on the bacteriostatic action of five per cent sulphathiazole in thirteen different ointment bases. They employed blood agar plates which were closely streaked with staphylococcus aureus and beta hæmolytic streptococci from a twenty-four hour broth culture. Slightly warmed ointment was applied on the plates by means of an eye dropper in amounts of 0·3 grams. After application of ointment the plates were incubated upside down at 37°C. The plates were examined after six, twelve, twenty-four and thirty-six hours. The width of the zone of the inhibition was measured with a millimeter rule under a hand lens. Similar ointments were made with sulphanilamide. The observations allowed these investigators to conclude that when sodium sulphathiazole is incorporated into suitable ointment bases it does not lose its bacteriostatic power. Sodium sulphathiazole is most rapidly effective when incorporated into bases of the water in oil type of emulsion which are rich in cholesterol, and it is likewise in these bases that the maximum effect was noted; and finally it appears that the relative mixture of fats and the emulsifiers in the fats of the base are of chief importance in liberating the incorporated drug.

Desoxycorticosterone Acetate for Epileptic Seizures*

McQuarrie and others (*J. Clin. Endocrinology*, 1942, June) determined the effects of desoxycorticosterone acetate on the water and electrolyte exchanges and on the occurrence of *grand mal* seizures in two severely epileptic patients. They found that this synthetic cortical adrenal hormone effectively antagonized not only the electrolyte responses, but also, under the conditions represented in these experiments, the seizure-provoking effect of the antidiuretic posterior pituitary hormone (pitressin). Preliminary therapeutic tests indicated that desoxycorticosterone decreases the tendency to spontaneous occurrence of *grand mal* seizures, also. The results presented tended to confirm an earlier conclusion regarding the nature of the convulsive mechanism in genuine epilepsy. Possible dangers from prolonged, indiscriminate administration of desoxycorticosterone acetate contraindicate its use until further investigations now in progress have been completed.

Sublingual Testosterone Therapy of Male Eunuchoids*

Lisser and others (*J. Clin. Endocrinology*, 1942, June) reported the sublingual administration of testosterone propionate, methyl testosterone and free testosterone, each dissolved in propylene glycol so that 0·2 c.c. contained 5 mg., in daily doses ranging from 10 to 25 mg. Five typical eunuchoids whose condition previously had been improved and who had been successfully maintained by parenteral, implantation and/or oral administration of testosterone compounds, continued their improved states on sublingual testosterone. In only 1 of the 5 cases was the sublingual mode of therapy more economical in milligrams required than the peroral route. All the patients preferred swallowing tablets to dropping a solution under the tongue. Four hypogonadal patients who had not previously received any testosterone therapy derived striking benefits subjectively and objectively from the sublingual administration of testosterone compounds. Larger oral doses of methyl testosterone would have been required to accomplish equivalent results. Sublingually, free testosterone was more effective than either methyl testosterone or testosterone propionate.

SURGERY

Utilization of Vitamin C by Cancer Patients*

Minor and Ramirez (*Cancer Research*, 1942, July) studied the daily utilization of vitamin C in 13 patients (7 controls, one patient with localized cancer and 5 with metastatic cancer). They found an average utilization of 67 mg. for the controls, 68 mg. for the patient with localized cancer and 125 mg. for the 5 with metastatic cancer. The natural occurrence of vitamin C in high concentration in actively growing and metabolizing tissues, and the probable function of this vitamin as a respiratory enzyme, suggest, these investigators believe, that this finding may result from an accelerated usage of vitamin C by carcinomatous tissue. If the presence of an active carcinogen in cancer patients is postulated an interference with cellular respiration by this agent might cause a compensatory accumulation of ascorbic acid in the affected tissues and thus diminish the excretion of vitamin C.

Essential Biochemical Derangements in Hyperthyroidism*

Barlett (*Arch. Surg.*, 1942, July) reports on the study of the total titratable acid in the urine in some 15 hospitalized patients with hyperthyroidism who were given diets, constant, in each case in the quantities of basic and acid forming components and in total daily caloric intake. These diets were maintained throughout the preoperative period of hospitalization and were ordinarily resumed on the third or fourth postoperative day. After a preliminary period of stabilization with the diet daily collection of twenty-four hour urine under standard precautions was carried out and was resumed in the postoperative period until the patient's discharge from the hospital.

As the result of his observations, he concludes that the essential biochemical derangement of thyrotoxicosis is an acidosis which is probably attributable in part to ketosis. The carbon-dioxide combining power of the plasma is maintained within normal limits even in severe exacerbations so long as the patient is at rest and his physiologic needs for water and nourishment are met, but a prompt decrease in alkali reserve occurs on exertion. During improvement on treatment preliminary to thyroidectomy and subsequent to operation, a profound fall in the total titratable acid of the urine, of the organic acids and of ammonia occurs, and the pH of the urine rises if the patient's intake of food is kept constant quantitatively and qualitatively.

In nearly 50 per cent of the patients who show prompt improvement after thyroidectomy a rise in the alkali reserve of the plasma occurs. He believes that acidosis bears a fundamental relation, probably causative, to the tendency of the patient with hyperthyroidism to water retention by the tissues through impairment of capillary integrity.

Eosinophilic or Solitary Granuloma of Bone*

Green and Farber (*J. Bone & Jt. Surg.*, 1942, July) report on the study of ten cases of benign destructive lesions of bone, either single or multiple which appeared to be identical with what has been described in the recent literature as "eosinophilic or solitary granuloma" of bone. All of the cases occurred in children under twelve years of age. Rœntgenographically, single lesions simulated bone cyst, osteomyelitis, or malignancy. Multiple lesions suggested multiple myeloma or malignancy, but in the rœntgenograms were indistinguishable from Hand-Schuller-Christian disease. There was little general illness associated with the disease in most instances. The symptoms were attributable mainly to the local process. The histopathology presented variations from "eosinophilic granuloma" to "lipogranuloma," which is interpreted as identical with certain stages of Hand-Schuller-Christian disease.

Ordinarily, the lesions have healed quite promptly after roentgen radiation, and, after curettage. Nine of the ten patients were well on a follow-up examination three to ten years after they were first observed. One had died, the details of which case are unknown. As suggested by one of the authors in a previous communication, eosinophilic or solitary granuloma is not a distinct new entity, but a variant of the basic process of which the clinical pictures known as Hand-Schuller-Christian and Letterer-Siwe diseases are other examples. Despite the excellent prognosis in nine of the ten patients in this group, in which the recognized lesions were restricted to bone, if the suggested relationship to Hand-Schuller-Christian disease exists, the prognosis given in an individual case should be guarded and the possibility of visceral lesions should be kept in mind.

Treatment of Chancroid with Sulphathiazole and Sulphanilamide

Korublith and others (*J.A.M.A.*, 1941:117:2150) reports on the results of treatment with sulphanilamide and sulphathiazole of 175 consecutive ambulatory patients with chancroid infection proved by positive intradermal reaction to Ducrey vaccine and by identification of the Ducrey bacillus on smears stained by the Unna-Papenheim method (methyl green pyronine) which was found to be the most important single diagnostic criterion of chancroidal infection. Sulphanilamide was given in 150 patients and sulphathiazole in 25 patients; sulphanilamide was given orally for 14 days; a total of 45·6 gm. was found adequate. Sulphathiazole was given for 10 days and a total of 20 gm. sufficed. Local use of sulphanilamide powder may be of use. All the cases treated with chemotherapy were cured. Reinfections occurred in 5 cases and there was 1 case of questionable relapse, all these cases healed promptly on the institution of a second course of treatment. Surgical measures of any description were contraindicated and found unnecessary. Aspiration of large inguinal abscesses was found sufficient in some cases; spontaneous absorption of small abscesses took place in 5 cases during chemotherapy. The author maintains that sulphanilamide therapy may be used as a therapeutic test in differential diagnosis of chancroid.

Sulphadiazine in Burns

Pickrell (*Bull. Johns Hopkins Hosp.*, 1941:69:217) from the Department of Surgery, Johns Hopkins Hospital, in a preliminary report records the results obtained in the treatment of 115 cases of burns with 3 per cent sulphadiazine in 8 per cent triethanolamine. The results were so satisfactory that this treatment is now followed in the hospital in every case of burn. The local treatment is as follows:

The patient is placed on sterile sheets and treatment for shock is immediately started. While the surgeon is scrubbing up, the nurse sprays the burnt areas with sulphadiazine mixture. The burnt areas are not washed or cleansed in any other way. Spraying is continued every hour on the first day, every two hours on the second day, every three hours on the third and every four hours on the fourth day. The author points out that sulphadiazine can be safely used in and around the eyes with impunity. (It can be detected in the blood within some hours after having been sprayed on the surface of the burn).

Tocopherols (Vitamin E) in the Treatment of Primary Fibrositis

Steinberg (*J. Bone & Jt. Surg.*, 1942:24:411) writes that primary fibrositis which is uncommon in the young people but common in the fifth decade, being equally divided between the sexes. It is characterised clinically by severe pain and tenderness which may be localised or generalized, the localized areas being not only tender but usually indurated and under spasm and generalised condition being usually associated with generalised stiffness. Drafts and cold, damp weather definitely

aggravate the condition or initiate an attack; warm weather or application of local heat brings relief. Fibrositis is usually characterized by the absence of an increased sedimentation rate, leukocytosis or fever. Histologically it showed marked proliferation of the fibrous tissue and œdema and many fibroblasts and blood vessels with many new capillaries without any leukocytic reaction.

A large number of cases of primary fibrositis were treated with tocopherols by either oral or parenteral routes—in both cases the drug was found to be very efficacious. There is the striking similarity between the pathological changes of primary fibrositis and nutritional dystrophy and this is no mere accident. The author maintains that this fact plus the marked clinical improvement obtained in both conditions by giving tocopherols indicate that primary fibrositis is a metabolic disorder concerned with deprivation of vitamin E. The dosage recommended is 300 milligrams of mixed tocopherols daily for the first week and 150 milligrams by mouth daily for the next two weeks. A maintenance dose of 100 milligrams daily may be continued indefinitely. The calculated maintenance requirement of 1 milligram of tocopherols per kilo of body weight should suffice for the average individual.

Oral Sepsis and Eye Affections

Rooper-Bell (*Brit. J. Ophth.*, 1942:26:141) in his Richard Memorial Postgraduate Lecture at Birmingham stressed on the importance of oral sepsis in relation to affection of the eye. Transmission of sepsis is easy on account of the close connection between the nerves, lymphatics and blood vessels of the mouth and eyes. The eye symptoms observed in young women and at times attributed to early disseminated sclerosis, have been shown to be due in many cases to oral sepsis and dental treatment clears up the condition. Blinking and blepharospasm result from dental irritation and the onset of squint in infants is associated often with teething. Congestion and inflammation in fifth nerve area are causal factors in primary glaucoma and many severe eye conditions may have their origin in dental lesions, even when of an apparently trivial nature. Early diagnosis of dental sepsis whether caused by buccal ulcers, dead teeth, septic pockets or even ill-fitting dentures is of great importance and is of immense help to an effective treatment of all eye conditions.

OBSTETRICS AND GYNAECOLOGY

Treatment of Tetany in Pregnancy

Anderson and others (*Am. J. Obst. & Gynec.*, 1942:43:547) give in the following lines the summary of their observations:

A review of 240 cases of tetany in pregnancy, occurring in the previous literature has been presented. Classification of the cases into 4 groups was attempted. 26 cases were of the postoperative hypoparathyroid type of tetany with pregnancy; 145 were of the indiopathic or spontaneous type, including in the group those cases which occurred during the pregnancy, during delivery or in the postpartum period. The third type consisted of a dietary type due to low calcium intakes and vitamin D deficiencies of which 9 cases were reviewed. The last type included 60 cases of tetany associated with thyroid deficiency.

The earlier literature stresses the relationship of female sex organs and pregnancy in the development of tetany. Other more specific factors, such as menstruation, lactation, blood loss at the time of delivery, hyperventilation and low calcium intakes have been considered.

Formerly tetany in pregnancy was considered an indication for therapeutic abortion, because of the high foetal mortality rate, also the tendency of newborn infants to develop tetany and a definite maternal mortality rate.

During the past decade there is a much better prognosis for both the pregnant mother and her baby. A combination of oral and intervenous use of calcium preparations, parathormone and dihydrotachysterol (A.T.10) carefully regulated by the patient and her physician yielded successful results in 12 of 13 cases in the literature. No abnormal alterations of the labours or delivery were observed. Careful observations of mothers and babies revealed no unusual abnormalities. Concurrent toxæmias must be watched for in cases of tetany in pregnancy as pre-eclampsia occurred in a few cases reviewed in the series.

Present Status of Treatment of Gynaecological Cancer

Kamperman (*Surg. Gynec. & Obst.*, 1941:72:384) reviews 485 cases of gynæcological cancer treated in Harper Hospital, Detroit, from 1922 to 1935 and discusses on the basis of 5- and 10-year survival curves and other statistical figures, the more salient points which have left their landmarks on the routine clinical procedures, thus leading to the present day status in the treatment of gynæcological cancer. Of the 835 cases reviewed 636 were cervical carcinoma, 77 carcinoma of the fundus uteri, 171 carcinoma of the ovary, and 31 carcinoma of the external genitals. All fatalities from whatever cause and all untraced cases were regarded as cancer deaths.

For carcinoma of the cervix irradiation has proved to be the method of choice and in recent years operation has been performed only in rare instances. As the chief reliance has come to be placed upon radium applied in the largest tolerable doses, supplemented by supravoltage therapy, the 5-year cure rate has reached an average of 35 per cent as compared with 16 per cent in the earlier years.

In carcinoma of the fundus a combination of surgery and irradiation has been used whenever possible. With increase in radiation dosage the average 5-year survival period rose from 30 per cent prior to 1930 to nearly 50 per cent after 1933.

Ovarian carcinoma was treated for the most part by combination of surgery and irradiation. The use of supravoltage therapy in this group seems to have had no effect on the 5-year and 10-year survival rate which averaged 20 and 18 per cent respectively throughout the period of the survey.

In carcinoma of the external genitals treatment was highly individualized, surgical procedures being common for lesions about the vulva, with irradiation frequently the sole procedure for carcinoma of the labia and vaginal canal. The 5-year survival rate was 16 per cent; none of the patients with vaginal carcinoma lived as long as this.

The advantage of the supravoltage therapy is attributed to the following 3 factors: (1) there is a greater differential action of the radiation with increasing voltages, permitting the administration of a proportionately large total dose, (2) there is an increase in the total energy which can be delivered to obtain the

same skin erythema; (3) there is an increase in the depth dose, making possible a larger absorption at the site of deep seated lesions. Clinically this means that a larger total dose can be employed to carcinoma with less injury to the skin and the surrounding structures.

PEDIATRICS

Spastic Children

Bridgman (*Am. J. Dis. Child.*, 1942:64:11) on analysis of the study of 123 spastic children examined in the Children's Psychologic Clinic of the University of California Medical Centre points out the following important facts:

1. A history of prolonged labour is significantly more frequent for the spastic children studied than for the series of children born at the University of California Hospital. 2. Prolonged labour and instrumental birth are much more frequent in the histories of the first-born spastic children than in those of the total spastic group. 3. The frequency of the use of a forcep in delivery often after a short period of labour, is more than 4 times as great for the spastic children as for the hospital series. 4. A history of premature birth is much more frequent for the spastic group than for the children born in the hospital. 5. Abnormal presentation (breech, face, shoulder) was more frequent for the spastic group than for the hospital series of 500 cases. 6. Moulding of the child's head does not seem significantly related to the development of the spasticity. 7. More than half of the spastic children were first-born. 8. The intelligence of spastic children tends to decrease with increasing age.

Paraplegia due to Measles Encephalitis: Report of a Case

Ward (*Arch. Pediat.*, 1941:58:83) in reviewing 11,147 cases of measles observes that in 5 instances encephalitis occurred. In the case reported there was severe encephalitis and the child's condition remained very grave for several days. The encephalitic condition gradually subsided but it was soon followed by signs of a transverse myelitis with flaccid paraplegia and 'cord' bladder. Complete paralysis persisted for nine days after which recovery started and was complete in 2 months except for a sequela of spastic paralysis of the legs.

Sulphathiazole Therapy of Infantile Diarrhoea

Taylor (*J. Pediat.*, 1941:18:469) reports on the use of sulphathiazole in every second patient (13 in all) of the 27 children admitted to the Duke Hospital, in the initial oral doses of 1 gm. per year of age upto maximum of 3 gm. followed by the same daily amount divided into 6 doses. The average time required for the stools to reach four a day and of normal color and consistency was three and two-tenths days in sulphathiazole-treated group and fifteen and six-tenth days in the control group. There were no deaths in the sulphathiazole-treated group. Of the 14 controls 2 died, one after a protracted hospital course of 27 days and the other nine hours after admission.

Promine in the Treatment of Some Acute Infections

Toomey and Roach (*J. Pediat.*, 1941:18:1) report on the successful use of promine [a sodium salt of p.p'-diamino-diphenyl-sulfone-N, N'-(dextrose sulfonate)] in acute infectious diseases. The drug was used intravenously in 154 patients and this route was chosen because of the severe reactions caused by oral use and local pain caused by subcutaneous injections. Two groups of cases were treated: Group 1 included patients with follicular tonsillitis, herpes zoster, streptococcic infection of the respiratory tract, scarlet fever and its complications, bronchial and lobar pneumonia and cellulitis and 1 patient with erythema multiforme. In 34 instances the temperature dropped down to normal within 12 to 24 hours with concurrent improvement in the symptoms; in 24 cases the temperature came down by lysis within 48 hours and the improvement in the symptoms was noticed within 24 hours; 18 patients had fair responses and 16 had no benefit from the drug; most of those who did not get any benefit had a complication of scarlet fever—in all 82 per cent had definite clinical improvements from the drug. Group 2 consisted of 59 patients with erysipelas of whom 86 per cent were improved by the drug. Complications noted in using the drug were: rash 4 cases, cyanosis 10, nausea 9, vomiting 6, headache 2 and vertigo 1. The authors hold that grain for grain the drug is found less toxic than either sulphanilamide, or sulphapyridine; even when used in the presence of red cells in the urine caused by some infection, it did not aggravate the condition.

Some Difficulties in the Treatment of Nutritional Anaemia in Infants

Evans (*Med. World*, 1942:56:405) writes that nutritional anæmia is fairly common in young children. The treatment of such anæmia is simple in essence and consists of giving adequate doses of iron and getting the baby to take mixed diet suitable to his age. It must be remembered that deficiency diseases run together (rickets is a common associate of nutritional anæmia) and the diet as a whole must be rectified. It happens, however, that the results are not quite upto the mark in some cases and the causes of the unsatisfactory result are: (1) insufficient dosage of iron, (2) gastric achlorhydria, (3) alimentary disturbances usually diarrhœa interfering with the administration or absorption of iron, (4) infection and (5) lack of copper. Iron and ammonium citrate is usually well tolerated by children and a satisfactory dose is 15 grains a day, divided into three doses, given after meals. Gastric achlorhydria is common in anæmic children and it interferes with the abosorption of iron; 5 minims of dilute hydrochloric acid, thrice daily may be used with benefit; pepsin one grain may be added to it. Vomiting and diarrhœa are sometimes caused by iron and often made worse by it. In such cases it should be omitted for a short time and another perparation may be tried. The presence of even mild infection will interfere with the formation of hæmoglobin and as these children are prone to infection this is a fruitful source of trouble. Most anæmic infants get on well if iron alone is given but a few do not thrive until copper is added.

Abstracts marked with an asterisk are prepared by the American Medical Association and distributed by the Department of State of the United States Government—Editor, J.I.M.A.

BOOK REVIEWS

HAND BOOK OF APPLIED PHYSIOLOGY OF THE NERVOUS SYSTEM, THE SPECIAL SENSES, MUSCLE AND NERVE—By Dr. H. N. Das, M.B., M.D. 1942. Pages xvi+444. Demy 8 vo. Illustrated. Published by Das Gupta & Co., Calcutta. Price Rs. 6/-.

This book comes to students from the pen of an experienced professor connected with a medical college for a long time. From his close association with students he could exactly know their difficulties in learning maximum about a subject in minimum of time. When one goes through the pages of the book, it is apparent that he has been successful in his efforts. Every subject has been discussed thoroughly but concisely and clearly.

An additional feature of the book is the appendix where all the questions of different Indian universities are incorporated. With each question a guide has been given with reference to the page of the book where the subject has been dealt with, so that it becomes easier for a student to answer the question properly. We recommend this book to all students.

B. P. NEOGY.

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CORRESPONDENCE

The Editor is not responsible for any views expressed by contributors—EDITOR.

THE EDITOR, *Journal of the I. M. A.*, Calcutta.

MALARIA PROBLEM

Sir,

The admirable article by Ghosh & Roy on the "Quinine Problem in India" in No. 2, *Journal of the I.M.A.*, has apparently received the general appreciation it deserves. The *Madras Mail,* for instance, one of the most prominent newspapers in the South, has made it the theme of a rather impressive and generally consenting leader.

Yes, it is true, malaria constitutes to-day one of the most urgent problems of "Social Medicine", even more, it probably constitutes the most important among all the important problems in India.

Therefore, it is highly desirable that, apart from the official research institutes and big hospitals, also the entire bulk of medical men should develop more interest in the malaria problem, which is more than a mere quinine problem.

I think that the correspondence column of the Journal of the I.M.A. could represent the frame for an exchange of opinions regarding all problems, connected with malaria. Would it not be possible that the staff of this Journal could summarize the results, once the discussion may have come to an end, and communicate the suggestions received to the Central Government?

I am firmly convinced that such a discussion would bring to light quite a lot of most valuable items, although some unimportant statements and negligible particulars as well. Every doctor should participate who believes he has something to say.

The following is my humble contribution towards such a proposed discussion:

(i) *Prophylaxis*: Ghosh & Roy are perfectly right: Notwithstanding the importance of preventive measures, we should, under present conditions, not expect too much from prophylaxis. Curative measures are the main weapons in our antimalarial combat of to-day.

However, I wish to emphasize the value of one prophylactic measure, which as far as I can see, is not in general use in this country: prophylactic blood examinations. Regular blood tests within certain intervals, *e.g.*, once in a fortnight, permit an early diagnosis of malaria, sometimes long before the appearance of any clinical signs and symptoms.

If the disease is diagnosed before the patient is manifestly ill, it can easily be cured, by a minimum of quinine, without relapse.

Is it possible to make regular blood tests on a large scale? I cannot dwell on particulars here; anyway, I believe such blood examinations are not more difficult than prophylactic vaccinations and inoculations on a large scale, although a correct blood test (thick film, 5 different tests can easily be made on the same slide) may take a little more time than vaccinations or inoculation.

(ii) *Treatment and Quinine problem*: A considerable amount of quinine may be saved, if instead of oral application, especially instead of application of tablets, absorption of which is so often quite incomplete, more and more quinine injections would be given. By means of systematic intramuscular treatment it should be possible, in a considerable number of cases, to achieve the same result with 10 gr. of quinine as with 20 gr. by oral use. Would it not be an interesting task for one or the other hospital to investigate, by comparison of cases treated by oral and intramuscular application, how much quinine can be saved by the latter method? By combining intramuscular quinine with intravenous arsenic treatment probably even more quinine may be saved.

The suggestions of Ghosh & Roy, that quinine should be reserved for treatment of malaria only, are certainly worth considering. However it does not seem to me likely that doctors are chiefly responsible for any waste of quinine. What a large amount of quinine is being consumed daily by the public, without medical advice, against every common cold and every supposed, in fact non-existent, attack of malaria! What I said of quinine counts for the synthetic antimalarials as well. Only a couple of days ago I saw a young girl of 14, who was treated by her mother with Atebrin, 10 gr. a day, (!) for 3 days. She did not show any sign of malaria, whatsoever, but developed a very severe jaundice, whether due to overdosage of Atebrin or not, I cannot say. At any rate, treatment with that rare and costly drug was highly objectionable in that case.

I suggest that quinine, for the time being, should be available to laymen on receipt of a medical prescription only, under the same restrictions as morphine and cocaine.

(iii) *Substitutes*: There can be little doubt that indigenous medicine is familiar with a good many antimalarials, which I do hope, will be investigated before long, if necessary with the aid of practitioners of indigenous systems.

May I report, by the way, that eucalyptus oil as well as decoctions of eucalyptus leaves have been used as antimalarials also in western medicine, especially during the 19th, but even at the beginning of the 20th century.

I am, by no means an expert in indigenous medicine, yet I beg to refer in this connection, to a substitute of quinine, use of which as an antimalarial is generally forgotten to-day.

The late German pharmacologist Hugo Schulz, in one of his outstanding textbooks, (Deutsche Arzneipflanzen, Leipzig, 1921, p. 237) mentions, as an antimalarial specific, the common chestnut. His reference seems to me important enough that I am finishing this letter by translating it herewith from the German text:

"The common chestnut, Aesculus hippocastanum, contains chiefly in the bark of the young branches, a glucoside, Aesculin, which presents itself as a white, crystalline powder of bitter taste.

"Aesculin has been highly recommended, during the last century, by French physicians for the treatment of malaria, and it was claimed to be effective, in doses of a gramme (15 gr.), even in cases when quinine failed. I do not know, how the matter stands, however, I think it worthwhile to resume the problem regarding the supposed and even probable action of Aesculin.

"If Aesculin really does, what it is claimed to do, we would have, in many cases, a cheap and easily available substitute for quinine." I am etc.,

Kodaikanal, S. India
Thomas Cottage,
22-11-42.

E. G. H. KOENIGSFELD